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THE
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JULY, 1907.

ORIGINAL ARTICLES.

HYPOPARATHYREOSIS, STATUS PARATHYREOPRIVUS, AND TRANSPLANTATION OF THE PARATHYROID GLANDS.

By W. S. Halsted, M.D.,
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We have no term except tetany to express the condition resulting from an insufficience of parathyroid tissue or parathyroid secretion. Probably the explanation of this is the fact that hitherto no instance of what might be termed hypoparathyreosis of a degree less than the tetanic one has been described. I would, therefore, propose Status Parathyreoprivus and Hypoparathyreosis as appellations to designate the state of the individual suffering from partial or complete loss of parathyroid tissue.

We have had the exceptional opportunity to observe for about fourteen months symptoms due to parathyroid extirpation or destruction and ameliorated at times by the administration of the parathyroids of beeves. Tetany has not been permitted to develop nor the manifestations of deprivation of parathyroid secretion to mature sufficiently for the production of the Trousseau or the Chvostek phenomena. We might define the condition of this patient as subtetanic hypoparathyreosis.

The first to describe in recognizable form the affection which we term tetany was a German named Steinheim, who in 1830 termed it Hitziger Gelenkrheumatismus, believing that the disease was closely allied to acute multiple articular rheumatism. The following year there appeared a publication by Dance entitled *Observation sur une espèce de tetanos intermittent.* This author recognized the essential feature of the affection, the intermittent character of the attacks of spasm.

Within the next few years numerous articles by French authors were published on contractures of the extremities, which, however, shed very little light on the subject. The especial contributions to the knowledge of this disease rendered by Trousseau, Erb, Chvostek, and other more recent writers are probably well known. In 1845, Trousseau, having observed the attacks in a nursing woman, termed the affection "contracture rhumatismale des nourrices." But in 1852 the disease was carefully studied by Lucien Corvisart, who proposed for it the name tetany, an appellation which it has since maintained.

Tetany has been observed as an endemic, an epidemic, and a so-called idiopathic form in apprentices of various forms of hand labor; in infections, including malaria; in intoxications; in gastrointestinal disturbances; after exposure, particularly in children; during teething; in Bright's disease; in pregnant, confined, and nursing women; as a symptom in certain affections of the nervous system; after operations for goitre.

In 52 extirpations of the thyroid gland in Billroth's clinic, tetany occurred 12 times, or in 23 per cent. of the cases; death occurred in 9 of these. Von Eiselsberg in 1890 stated that 30 cases of tetany due to thyroid extirpation had been observed. Of these, 7 are said to have recovered, 3 to have suffered chronically from the affection, and 13 to have died. The result in the remaining 7 cases was unknown. Of the 12 carefully observed cases of Billroth there was, probably, 1 recovery; 2 cases became chronic. A number of chronic cases of operative tetany have been observed, some of them for many years, by various surgeons; these patients are reported as having periodic attacks of tetany at irregular intervals, and as being quite well between these tetanic manifestations. Total extirpation of the thyroid yielded a much greater percentage of tetany in the hands of some operators than of others; and so before anything was known of the parathyroid glands total extirpations were rarely undertaken except for malignant disease. In several of these cases fatal tetany occurred.

The study of our case of aborted operative tetany has been greatly facilitated by the coöperation of the unusually intelligent patient, a cultivated woman who, prior to the operation, was in excellent health except for the distress of a large and rapidly growing goitre. On May 5, 1904, I excised, for this patient, at the Johns Hopkins Hospital, the left lobe of the thyroid, endeavoring to leave *in situ* both of the parathyroids of this side if possible. A careful examination of the removed lobe, by Dr. MacCallum as well as by myself, did not reveal a parathyroid gland. The patient returned March 26, 1906, to be relieved of the asphyxia and apprehension caused by the remaining lobe, which from the usual change in position following the unilateral operation, and from a very considerable increase in size, resembled, on superficial examination, a bilateral and rather symmetrical, large goitre. The patient having said to me before operation, "Remove it all if you can, for I would rather take thyroid extract the remainder of my life than have the goitre return," and as the goitre had grown rapidly since the previous operation I determined to remove as much of the thyroid proper as could, in our opinion, safely be done. Although the goitrous lobe was carved in a way which I thought would not endanger the lower parathyroid gland, the knife actually grazed the edge of this body, which I detected immediately on the excised portion of the right lobe, in quite a normal relation to a branch of the inferior thyroid artery. It happened that on the preceding day I had performed the same operation, and in much the same way, for a goitre of the right lobe of the same kind, on a patient whose left lobe I had removed seven years ago. A stump of the same shape and in the same position was left, but it contained, presumably, the right inferior parathyroid. In both patients the four thyroid arteries had been tied in the course of the two operations on each. We watched the convalescence of the patients carefully; the one first operated upon recovered without incident, but the second was not herself from the outset, although her temperature and pulse were about normal. "This recovery is entirely different from the previous one," she would frequently remark. "I do not understand myself. I have never felt so before." She was unnaturally flushed for two or three days; was restless and slept badly. But she believed herself to be improving until the seventh day, when queer feelings developed, so emphasized at times that she designated them as attacks, although she was uncomfortable and a little apprehensive between the attacks even at this time. It is interesting to note that even in the beginning, when the symptoms were very indefinite and mild, they became concentrated in a way to suggest periodic explosions.

My daily visit to the patient was omitted on the seventh day. On the following day, the eighth since the operation, she had, during the morning, several "attacks," and at my visit, about 2 P.M., I found her quite excited and not a little apprehensive. While attempting to describe her feelings in these attacks or "spells," there developed one, superinduced, it seemed, by the incidental increase in mental activity. It passed through phases precisely as she had described them for the previous attacks. "I feel a numbness in my lips as if the blood had stopped in them; a sort of wave of numbness is passing over my face;" and a few seconds later, "Now it is going into my arms; now it is passing off. Oh, it is dreadful! I would really rather die than have even a few more attacks like these. They are indescribably awful." On the disappearance of the "numbness" or lifeless feeling of the lips, for example, it seemed to the patient as if the blood which had suddenly been blocked was rapidly let into the affected parts again. We were unable to see variations in the appearance of the lips during the attack. The patient insisted that she could not have closed her eyelid without a finger if her life had depended upon it. This suggests hysteria, but those who saw her in this attack doubted not the existence of a very serious condition. She looked ill, quite alarmingly so. Dr. MacCallum, appealed to in haste, placed at our disposal his entire collection of the dried parathyroids of beeves. I was so concerned about the patient and so confident as to the nature of the symptoms that it did not occur to me to make the Chvostek and Trousseau tests until several parathyroids had been given, and even then I feared for several days to make prolonged pressure on the femoral artery, or to test the electrical reactions. The effect of parathyroid exhibition was almost instantaneous and most marvellous. Large doses were given at first, six dried glands every three hours.

Hoping to stimulate hypertrophy of any parathyroid tissue which might still be in the body we reduced the number, in the course of a few days, gradually to one gland three times daily.

The patient could not, for several weeks, bear further reduction in the dose of parathyroids, although she was perfectly willing and almost eager to attempt it, notwithstanding her feeble and strange, neurasthenic-like condition.

Tapping the facial nerve, repeatedly tried, has not once elicited a response. Nor has pressure of the femoral artery, exercised for periods of four and five minutes, been followed by contractions.

Naturally of a most cheerful and optimistic disposition, she made a brave fight during brief frequent, dispiriting periods, of which the duration was rarely more than a few hours—often only one, though occasionally twelve. Attempts to test electrical reactions distressed her so acutely that they were given up. Convergence tests of the internal recti muscles unnerved her; she involuntarily frowned when the finger approached the nose, as if the effort to follow it were actually painful. A slight, hard œdema of the legs between the knee and ankle and a cold skin persisted for weeks. For a brief period there was albumin in the urine. Sudden changes of weather, particularly to cold, affected her badly. On showery days she would say, "I feel precisely as if I were taking electricity." She expressed at the outset great aversion to sweets and a desire for acids, but when parathyroids had been given for perhaps two days she nibbled bonbons with relish.

For the eight days after operation and preceding the administration of the beeves' parathyroids there was a rapid loss of weight. After the administration of parathyroids was instituted there was for several months a slow gain in weight.

Our patient remained in the hospital for twelve (?) weeks after the operation, taking the fresh parathyroids of beeves at times in large amounts during the entire period, except for the first thirty-six hours after the development of the privation symptoms and on the few occasions when the dried glands were substituted for the fresh, either for the sake of comparison or because the supply of fresh parathyroids had been unexpectedly interrupted. Their exhibition was by mouth only, as prolonged hypodermic administration of the crushed glands could not, it seemed to us, be endured, owing to the physical properties of the emulsion, the bulkiness of the dose, and the annoyance and danger from the hardly avoidable infection. The patient promptly discovered, as did we, that the dried glands, a nauseous dose, seemed less effective than the fresh.

In preparation for ingestion the fresh glands were carefully freed from fat, chopped very fine, and served on thin, crisp water-crackers. The dried, powdered glands were usually given in gelatin capsules. During the summer and autumn of 1906 the patient, at home in the South, took the dried glands. Their discontinuance in November, 1906, for about three months was without effect, appreciable either by the patient or her family; and after resumption of the parathyroid feeding for about one month without benefit the ingestion of the dried preparation became so revolting that its exhibition was discontinued.

March, 1907. From correspondence I judge that the patient has in some ways slowly improved. There has been a little gain in appetite and in strength, and, as I have said, a considerable gain in weight. But she is an invalid. Her nervous system is quite wrecked. Conversation, even of a light and bright nature, unnerves her; so does a walk of one hundred or fifty or a less number of yards, or even sitting up in a chair for an hour or two. Shall we term this condition a cachexia parathyreopriva? And may not, in some cases, the cachexia thyreopriva, so called, be compounded of thyroid and parathyroid privation? Cachexia thyreopriva must be studied from a new point of view. Our patient writes that during the past three or four months pain in her knees, elbows, and shoulders is quite constant and at times distressing. There is, perhaps, no pain except that induced by motion, although this may persist for many minutes, particularly after some hasty, uncalculated movement. Exercise of the knees elicits crackling noises which at times may be heard by those at the bedside. The nature of this affection of the joints, or joints and tendons, has not been determined.

I have it in mind to make hypodermic injections of the parathyroid-nucleoproteid which Dr. Beebe, of New York, is now, he kindly tells me, experimenting with, and hope that the experiments in transplantation of the parathyroids in animals which I have been conducting since January may give results to encourage the therapeutic use of this measure in the case of our patient for whose pitiable plight I am responsible.

Picture the operator's state of mind if, instead of one, a number of cases of tetany or hypoparathyreosis were placed to his—shall I say, credit? A certain measure of consolation is found in the knowledge that the deplorable result was brought about in the effort, fortunately successful, to cure a hardly endurable condition, one which menaced life. The patient charitably insists that her present state is preferable to the wholly intolerable one from which the operation freed her.

THE TRANSPLANTATION OF PARATHYROID GLANDS. I had been seriously deliberating as to the advisability of attempting subperitoneal transplantation of the parathyroids for this patient when there appeared the impressive report of Payr[1] on the successful transplantation of portions of the thyroid gland into the spleen, both in animals and the human being. From Payr's concluding summary I abstract the following: The spleen possesses, thanks to its altogether peculiar and rich blood supply, unusually excellent conditions for the transplantation of all kinds of tissue. The circulation is rapidly restored, a relatively small proportion of the transplanted part is sacrificed to central necrosis, and the processes of regeneration are more active and more extensive than in any of the methods of transplantation heretofore employed. The chief consideration against the transplantation into the spleen is the danger of hemorrhage. But by numerous experiments in animals a technique has been developed which has effectually overcome this danger. Chief among the technical features are the use of the transplanted organ as a wedge in the splenic wound and of the omentum as a support for the stitches and a covering for the line of incision in the spleen. It has been possible by this method of splenic transplantation to keep alive for a period as long as three hundred days animals which otherwise would have perished with symptoms of insufficience of the thyroid gland. Into the spleen of a child, six years old, totally imbecile and with severe infantile myxœdema (treated almost without result for a period of three and a half years with thyroid feeding), a large piece of the thyroid gland of the perfectly healthy mother was transplanted. The result, to the present time, somatically as well as intellectually, is highly satisfactory.

Payr's work stimulated me to attempt splenic transplantations of the parathyroids on dogs with the hope that if successful I might do the same thing for my patient. As the result of a very considerable experience in thyroid transplantation gained by me in 1888 the spleen seemed an ideal organ for the purpose. The technique of the thyroid transplantation as developed by Payr was very simple. The block of thyroid tissue used as a wedge assisted in the arrest of hemorrhage from the spleen. But the tiny shavings of disks of parathyroid tissue could not play such a role, and at first I found the bleeding from the little holes made bluntly in the spleen annoying because it prevented the introduction of the parathyroid films, promptly floating them entangled in a clot to the surface. After a few unsuccessful trials the following procedure was devised: A split thread of china-bead silk is armed with two very fine needles, No. 16. The properly prepared parathyroid, or preferably the little supporting rim of thyroid gland, is perforated with one of the needles and the minute gland slipped an inch or two on to the thread, great care being exercised to avoid crushing it. A little hole is made in the spleen with a fine-pointed artery forceps, the blades of which are opened wide enough to admit the parathyroid between them. Both needles, first one and then the other, introduced between the blades of the artery forceps down to the apex or bottom of the hole made in the spleen, transfix the spleen and emerge on the opposite side a few millimeters apart. The thread ends are pulled upon until the transplanted piece is carried to the bottom of the hole prepared for it. The spleen is then darned very gently with a mattress stitch or two of this split china-bead silk on the fine needles carried from quite a distance on both sides across the base and middle of the hole, and finally one of the threads which have been held taut is divided close to the under surface of the spleen and withdrawn. This method serves infallibly to fix these tiny films in the desired position. The bleeding from extensive tears, crushings, or cuts may be controlled by this method of darning.

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[1] Transplantation der Schilddrüsengewebe in die Milz; experimentelle und klinische Beiträge, Langenbeck's Archiv, Band lxxx, p. 730, also Verhandlungen der Deutsche Gesellschaft for Chirurgie, 35te Congress, i, 155, and ii, 503.

As the threading of these very fine needles is difficult, the double arming of the silk may be avoided by a slight modification of the procedure, which we have used of late. A single needle, threaded, is passed from the under surface of the spleen upward into the apex of the transplantation hole, made in the manner described; then, impaling a parathyroid, it is carried back into the hole from which it has just emerged and thence through to the under surface of the spleen a few millimeters from its point of entrance.

In the dog the inferior parathyroids in preference to the superior have been selected by me for transplantation, because being partly embedded in the thyroid gland and thus splinted by it they may be shaved with a sharp cataract knife or fine scalpel, on the exposed surface. Under and about the parathyroid a little thyroid tissue should be left. Thus framed in thyroid tissue the tiny parathyroid disk can conveniently be manipulated and speared by the needle without injury.

The thyroid glands, particularly when hypertrophied, as in Graves' disease, should serve admirably for transplantation of the parathyroids. On several occasions, operating for exophthalmic goitre, I have transplanted back into the other lobe or into the isthmus a parathyroid whose nutrition was cut off or imperiled. Once the parathyroid so transplanted had been unintentionally excised. In a case of carcinoma of the stomach with gastric tetany and in the course of an operation for gastro-enterostomy, I attempted transplantation into the spleen of fresh parathyroids, obtained by Dr. MacCallum and Dr. Sowers from a body dead only about an hour. Unfortunately at the instrument table a drop or two of a solution of carbolic acid accidentally came in contact with these parathyroids, and hence I desisted from the attempt to transplant them, although the spleen was in my hand and already prepared for the transplantation.

I had the pleasure of demonstrating a few months ago at my clinic, and before the Interurban Clinical Club, which complimented me by attending it, microscopic sections showing the morphologically successful transplantation in dogs of parathyroids into the thyroid gland, and I hope to be able to report soon the result of functional tests.

We have transplanted parathyroids into the thyroid gland and spleen in 17 dogs. On each of two occasions as many as 8 dogs were used in the search for a large number of suitable parathyroids for splenic transplantation. In the spleen of each of 3 dogs as many as 7 parathyroids were at one time transplanted.

Tetany, inexplicable so recently as eighteen years ago, has been made comprehensible by one of the greatest and most impressive physiological discoveries of modern times. Four or more tiny bodies, so small as to have attracted no serious attention until, in 1880, Sandstrom classified them, quickly assumed upon the epoch-making discovery of Gley, in 1891, an importance hardly to be overestimated. Their situation, so deep in the neck, so carefully tucked away under the jaw and behind the thyroid lobes; their nutrition, each gland with a blood supply independent of the other and in no danger of cut off except by the surgeon's knife; and their number, generally four or more, might be viewed to indicate their preciousness. Their blood supply is a rich one and may well be subject on occasion to great and sudden increase, as in the active thyroid hypertrophies and hyperemias. Their vascular supply is so intimately related to and dependent upon that of the thyroid that they could hardly escape, it would seem, promptly and altogether, the effect of a sudden turgescence of the thyroid lobes, although controlled undoubtedly by regulating vital processes. The possibility of an interdependence of function between the thyroid and parathyroid glands has suggested itself to several investigators.

In this connection the "Note during Proofreading," appended to Dr. Reid Hunt's article on "The Influence of Thyroid Feeding upon Poisoning by Acetonitrile," is very interesting: "Later experiments show that feeding with parathyroids does not decrease the susceptibility of mice to acetonitrile; on the contrary, it causes a distinct increase of the susceptibility to this poison. Whether this increase of susceptibility is greater than that caused by an equivalent amount of proteid has not been definitely determined, but the indications are to this effect."

In Lusena's hands after experimental parathyroidectomy the average duration of life was only half as long as after parathyroidectomy plus thyroidectomy.

In this connection certain experiments on dogs made in 1888, and before we knew that the parathyroids had any function whatever, may be in point; in the light of our present knowledge they furnish interesting material for speculation. In a few instances[2] I

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[2] W. S. Halsted, An Experimental Study of the Thyroid Gland of Dogs, with Especial Consideration of Hypertrophy of this Gland, Johns Hopkins Hospital Reports, vol. i, Baltimore, 1896, p. 378.

Partial Isolation of Both Thyroid Lobes. The large branch of the thyroid artery which goes to the upper pole and the vein from the lower pole are not ligated; otherwise both thyroid lobes are completely isolated.

Example 1. Dog 104.

April 19, 1889. Open wound.

April 28. The dog seems perfectly well. The wound is healed.

April 30. The dog has tongue tremors and makes the licking movements with the tongue which we have frequently observed in dogs whose thyroids have been extirpated.

May 1. The tongue tremors are less pronounced. The licking movements are still vigorous.

May 7. The tongue tremors are pronounced. The dog has no general spasms.

May 14. The tongue tremors are very faint.

May 28. Dog seems perfectly well.

June 24. Hair is falling out, particularly over the eyes.

June 25. The dog has escaped.

June 27. Returned of his own accord. The hair is very thin all over his body. He scratches himself constantly. The œdema caused by the scratching gives to the skin about the face and head somewhat the appearance of myxœdema.

July 10. Has lost most of his hair. He is very anemic. His blood is used for direct transfusion into dog 109.

Autopsy. Both thyroid lobes are large and hard and very firmly bound down to the surrounding tissues by adhesions. With the microscope they were found to be hypertrophied. I believe, judging from the next example (dog 103), that this dog would have died ultimately with the symptoms of thyroid extirpation.

Example 2. Dog 103.

April 19, 1889. The wound is left open. The dog was profoundly depressed after the operation.

April 27. There is conjunctivitis of both eyes. The wound is doing well.

April 28. The dog has symptoms which are characteristic of extirpation of both thyroid lobes. He shivers, has tongue tremors, and general tonicoclonic spasms. His gums are inflamed. The membranæ nictitantes are conspicuous. He groans with each expiration.

April 30. The tongue tremors persist. The general spasms have ceased. The conjunctivitis is more pronounced. There is a very offensive gingivitis. Dog makes peculiar, almost incessant licking movements with his tongue. These peculiar licking movements have been frequently observed by me in dogs whose thyroid glands have been extirpated.

May 1. Seems better. He still has tongue tremors.

May 3. His hind legs are slightly stiff. The tongue tremors are scarcely perceptible.

May 7. The tongue tremors are more distinct. The conjunctivitis has almost subsided.

May 14. The tongue tremors are very faint. There is no conjunctivitis.

May 19. All the symptoms of extirpation of both thyroid lobes have reappeared.

May 21. The dog seems much better. The tongue tremors are very faint. There are no other symptoms. There is a slight subconjunctival injection.

May 23. Except for the faint tongue tremors the dog seems fairly well. He eats well, but is very thin.

May 24. Has no tongue tremors, but strong clonic spasms of the flexor muscles of all of his legs. The spasms of the temporal and ear muscles are especially pronounced.

May 25. Dog has no spasms and is much better.

June 14. All the symptoms of double extirpation have reappeared.

June 15. A.M. Dog found dead.

Autopsy. The left thyroid weighs 3 gms. and is very hard. The right thyroid weighs 1 gm. For the microscopic appearance of both lobes see Plate X, Fig. 7. The fatal result in this and the preceding case (104) is to be attributed probably to extensive destruction of the glands by blood extravasation. Otherwise we could not regard the changes which we designate as hyperligated all the vessels, veins and arteries of both lobes, except the superior thyroid artery and the inferior thyroid vein. In these cases the dogs ultimately died, notwithstanding the fact that as a result of the operation the thyroid lobes enlarged and microscopically showed the hypertrophic picture. These cases puzzled me greatly in those days, when tetany was believed to represent acute thyroid privation, for a modified and chronic tetany occurred, notwithstanding the hypertrophy of both thyroid lobes. Dogs so treated lived nearly three months on the average, in contrast to those with thyroid extirpation, which died usually in from two to nineteen days after operation. These are perhaps the first recorded cases of chronic experimental tetany. Both superior parathyroids were probably deprived of their circulation by the ligature of the superior vein plus the handling incident to the ligation, but the inferior or subcapsular glands should have had the benefit of a blood supply which was sufficient, not merely to prevent necrosis of the thyroid lobes but to cause their hypertrophy—hypertrophy confirmed microscopically. Hence there should have survived for each dog, it would seem, the two internal parathyroids as well as the hypertrophied thyroids. If the existence of antagonism between thyroids and parathyroids were established we might assume that the hypertrophied thyroids were imperfectly equilibrated by the two parathyroids, and that if thyroid hypertrophy had not resulted the two inferior or subcapsular parathyroids would have been more than ample for the prevention of tetany. This is the merest speculation and indulged in to call attention to the experiments which are, I believe, unique and might some day be repeated. In such case the interest would concentrate in the condition of the inferior parathyroids. With no parathyroid tissue remaining the dogs could not have lived, as they did, for months. The open wound and consequent suppuration in our cases may have partially destroyed the parathyroids. The cicatricial contraction may have been a factor plus the infection.

Of particular interest to the surgeon is that form of tetany caused by operation upon the thyroid gland. When total excision of the thyroid was practised for the cure of ordinary colloid and other goitres surgical tetany was comparatively common; then for nearly twenty-five years little was heard of this form of tetany, but now again it is making its appearance with alarming frequency and especially at the hands of the most experienced operators, those who are convinced of the advisability of removing in Graves' disease as much of the thyroid as can safely be done. The distinguished surgeon who was perhaps the first to warn against total excision of the thyroid gland is now among the first to advise for the cure of exophthalmic

[illegible] as very highly compensatory, for, notwithstanding the fact that the glands weighed two or three times as much as normal and showed the most advanced stage of hypertrophy, one dog died with symptoms of total extirpation and the other had the symptoms which I have described (dog 104), and would probably have died of thyroid privation.

goitre an excision, almost total, of this organ. Within a year or a little more, and in his latest 50 operations this operator has produced 7 cases of tetany; in none of these was the disease fatal, however; although in none have parathyroids in any form been given.

That operative tetany has of late so frequently resulted after a quarter century's interval of freedom from this complication of thyroid removal, is due to the facts that the operation for exophthalmic goitre is becoming much more frequent; that the proportionate amount of the gland removed in this operation is increasing, and that the loss of parathyroid tissue seems to be less well sustained by patients suffering from Graves' disease than by those afflicted with the hypothyreotic or colloid forms of goitre.

THE OPERATION. The development of the operation for exophthalmic goitre is an interesting story. Hemorrhage from the large, thin-walled veins was quite terrifying to the surgeon in the early days of the operation, and only a few weeks ago I heard a brilliant operator at work on a serious case exclaim: "This is like hunting for big game; instinctively, I look over my shoulder for a tree when I tackle a difficult exophthalmic goitre." The mortality from the toxemia of absorption, of blood and of thyroid secretion, was very high. And now the surgery of exophthalmic goitre centres in the problem of removing the greater portion of the thyroid gland without sacrifice of the parathyroids. I know from correspondence with the surgeons abroad who have especially devoted themselves to thyroid surgery that with them this problem is not solved.

I shall take it for granted that the situation of the parathyroid glands is fairly well understood; but a composite chart based on sixty-seven dissections, which were very kindly made for me by Dr. MacCallum, is of interest. The parathyroid bodies vary greatly in size and not a little in situation. The study of the situation of these little bodies in Dr. MacCallum's dissections and the diagrams based thereon and in the cases operated upon by myself, had given me the impression that much the densest grouping of parathyroids, in the composite diagram, would be about the insertion of the terminal branches of the inferior thyroid vessels, and that in comparatively few instances would these bodies have to be represented above or even as high as the tip of the superior pole of the thyroid. From our study, however, it was found that one is likely to encounter these little bodies, usually two on each side, at any level from the superior to the inferior pole on the postero-internal surface of the gland, but most commonly just internal to the rounded postero-external border and quite regularly near the site of the distribution of the terminal branches of the inferior thyroid artery. If the thyroid gland is lobulated in this situation, as is quite commonly the case, a parathyroid may be concealed in a cleft between the lobules. Not infrequently one of these epithelial bodies is located below the inferior pole of the thyroid gland and occasionally behind the sternum.

May the parathyroids be identified with the naked eye in the course of an operation? Yes, and almost infallibly; but perhaps not from any description that I can give. They are little ovoid, spheroid, lenticular, or very flat bodies, exhibiting much variety both in form and size and even in color. Externally they often resemble fat very closely in consistence as well as in color. On section, however, the fat about the deep cervical vessels and in the neighborhood of the thyroid gland is usually much whiter than the parathyroid, and the latter has a definite but exceedingly fine, almost microscopic, granular appearance. Differentiation from accessory thyroids or detached or partially detached lobulettes of normal thyroid tissue, is rarely difficult, although careful comparison of the suspected bodies with the tissue of the goitre may be necessary to convince one as to their nature. Partially or wholly detached portions of certain yellowish goitres, or adenomas of the thyroid, may bear a troublesome resemblance to the parathyroids. Lymphatic tissue is quite readily differentiated by the expert. Differentiation may be impossible if the parathyroids have been subjected to trauma, ever so slight.

The cause of tetany at the hands of well-versed operators is, I believe, less often removal of the parathyroids than interference with their circulation; and I am inclined to feel that the procedure of stripping the posterior capsule, so beautifully executed by Dr. C. H. Mayo, does not guarantee the safety of the parathyroid bodies. To assist in determining this point the removal of one lobe furnishes really no evidence whatever. It is only from the results of operations on both lobes that we can base an opinion. But who, in order to decide the question, will take the risk of causing tetany? I fear to do an operation, even on one side, except in very ill patients, which does not contemplate the leaving of a slice of the thyroid gland in the region of the ultimate distribution of the inferior thyroid artery. I avoid also ligation of the trunk of the superior artery, for fear of injuring the tiny branch to the superior parathyroid, and hence usually slice through the gland a little below the superior pole.

THE INTERPRETATION OF THE PULSATIONS IN THE JUGULAR VEINS.

By James MacKenzie, M.D.,
of Burnley, England.

Although the venous pulse has been recognized by clinicians for over two hundred years, the exact significance of its meaning today is not quite clear. It is only since graphic records have been taken of the movements in the veins that definite data have been obtained from which conclusions could be drawn. It was in 1863 that Bamberger and Marey published tracings of the venous pulse, being followed shortly by Geigel and Friedreich, but these tracings were taken independently of any timing with a standard movement. In 1868 Potain obtained tracings of the venous pulse simultaneously with the carotid artery, and after him numerous other observers, notably, Riegel, Francois-Franck, and Gerhardt investigated this subject. The general conclusion arrived at was that there were two kinds of venous pulse: one called the *normal* or *negative* venous pulse, the other the *pathological* or *positive* venous pulse. Generally speaking, this discrimination has prevailed in the literature of this subject.

In 1893,[1] I drew attention to the fact that the varieties of the venous pulse could not be satisfactorily included in these two simple forms, and that clinicians had entirely failed to appreciate the meaning and significance of the various forms of the venous pulse. The terms employed were utterly misleading: the so-called normal venous pulse was often as abnormal as the pathological venous pulse; the negative venous pulse was often largely composed of positive elements, and the positive venous pulse might often show negative evidence. As I had found that the presence of the auricular wave was a constant phenomenon in the majority of cases, I suggested that the term *auricular venous pulse* should be applied to these forms of pulsation in which the auricle occupied its normal place in the cardiac cycle, although in many cases evidence of the effects of the ventricular systole was present as well. On the other hand, I found that what had hitherto been called the positive venous pulse did not show any evidence of the auricular systole at its normal period in the cardiac cycle, and that the whole of the wave occupied the period of ventricular systole; I, therefore, employed the term *ventricular venous pulse.* Further, the appearance of the ventricular venous pulse implies a profound change in the rhythm of the heart, inasmuch as it is the evidence that the heart's contraction starts no longer at the mouth of the great veins.

VARIABILITY IN THE APPEARANCE OF THE VENOUS PULSE. To a great extent we are even at this day quite at a loss to explain all the conditions that give rise to a venous pulse. Some people in apparent good health show it, while in cases of marked heart failure it is entirely absent. Some people when in robust health show no signs of it, but if they become slightly debilitated the venous pulse may appear in the neck. In some cases of pernicious anemia the venous pulse may be a very prominent symptom, in others it may never appear. Some women during pregnancy develop a large venous pulse, others develop it only during the puerperium, while others never show the slightest sign of it. In irregular action of the heart it may assume, in certain cases, enormous proportions; in other cases there is not the slightest sign. A man may show it during one attack of heart failure, and during another and more grievous attack may show no sign of it. When one considers the foregoing variabilities in the appearance of the jugular pulse one is compelled to inquire more closely into the mechanism by which it is produced, and it will be found that our knowledge of the heart's action is even at the present day too slight to enable us to speak with certainty.

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[1] The Venous and Liver Pulses, etc., Jour. Path. and Bact., 1894, ii. In this article a full bibliography is given up to 1893.

CARDIAC FACTORS CONCERNED IN THE PRODUCTION OF THE VENOUS PULSE. In order to appreciate fully all the factors concerned it is necessary that we should recognize the very valuable discoveries of Gaskell on the functions, and some observations of Keith on the structure, of the heart. Keith[2] has shown that the musculature of the heart is built up of bands of muscle fibers, having as distinct places of origin and insertion as skeletal muscles, and that each bundle has its function as peculiar to itself as a skeletal muscle. But the heart-muscle fibers are distinct from skeletal muscle in this respect, that according to their special duties certain portions have individual functions more developed than other portions.

The work of Gaskell[3] has shown that the contraction of the muscle fibers is due to the possession by these fibers of certain very definite functions: the functions of rhythmicity, excitability, conductivity, contractility, and tonicity. All the fibers of the heart are not equally endowed with the same functions, some fibers being naturally endowed with those functions which are essential to the playing of their part in the mechanism of the heart. Thus some fibers have the power of contractility more developed than others, for it is evident that the muscle fibers constituting the apex of the heart must have different functions to perform than those that support the auriculo-ventricular valves. In the latter case the function of tonicity is naturally that one that would subserve the purpose of these fibers. It follows, then, that if the muscle fibers that prevent the regurgitation of the blood from the right heart into the great veins have their tonicity affected, they cannot effectively assist in closing the caval and tricuspid orifices. Keith has shown that this regurgitation during auricular systole is prevented by the contraction of a band of muscle in the auricular wall (the tænia terminalis), which shuts off the auricle from the superior vena cava. In some of the hearts I have sent him he has found these fibers elongated and attenuated, and during life there was great regurgitation of the blood into the veins during the heart's contraction. In like manner the fibers that support the tricuspid valves are very apt to lose their tonicity, and as these valves even in health are barely sufficient to close the tricuspid orifice, the slightest depression of the tonicity of the muscle fibers at once permits regurgitation from ventricle to auricle.

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[2] The Evolution and Action of Certain Muscular Structures of the Heart, The Lancet, February 27, 1904.

[3] The Contraction of Cardiac Muscle, in Schäfer's Text-book of Physiology.

TRICUSPID REGURGITATION. When I had taken tracings of the venous pulse and considered all the possible factors in its production, I took into consideration the fact that tricuspid regurgitation might play a very important part in the pulsation of the veins. Although its effects had been recognized in producing the so-called pathological venous pulse, no attempt had been made to show the connection between the so-called normal and the pathological venous pulses. That tricuspid regurgitation was the cause of the latter form was recognized, but no one had worked out how this tricuspid regurgitation began and gradually increased. Watching many cases and observing the signs of tricuspid regurgitation during life, and examining the hearts after death, I found abundant evidence that tricuspid regurgitation does modify even the so-called normal venous pulse. That my views were accepted by my colleagues in Britain was due to the fact that tricuspid incompetency is looked upon as so common, and so readily set up, that the whole train of my reasoning was conformable to their experience. But to my surprise I found that outside the British Isles my explanation was not accepted, as the fact that tricuspid incompetence was so easily set up had never been recognized.

Clinicians one hundred years ago, and John Hunter before them, recognized this tendency to incompetency of the tricuspid valves, but it was left to Wilkinson King,[4] in 1837, to demonstrate the relative natural incompetency of the tricuspid valves by a series of careful dissections and experiments. For several generations this has been the teaching in British schools, and in such text-books as those of Bramwell and Gibson the natural tendency to tricuspid incompetence is duly recognized. On the other hand, Babcock expresses surprise that Gibson should describe tricuspid regurgitation as a frequent occurrence, and, as an additional evidence of American opinion, Preble[5] recently bears testimony that tricuspid regurgitation is rarely recognized, and in citing some cases of pernicious anemia, he describes as new what has been taught in our schools for generations. Writers like Riegel, Krehl, Sahli, and others recognize tricuspid regurgitation when it is secondary to failure of the left heart, and in the extreme form when it produces the pathological or positive venous pulse. I have again carefully gone into the whole matter and made a series of new observations, noting the symptoms during life (particularly a murmur, systolic in time, heard loudest over the middle of the sternum, and over the third and fourth left costal cartilages), and I have followed certain patients until their death, and examined their hearts and found marked dilatation of the tricuspid orifice. But as even these cases might not be considered free from doubt, inasmuch as it could be held that the tricuspid dilatation was due to the changes taking place immediately preceding death, I have carefully noted the symptoms during life in patients whose tricuspid valves had been damaged by old-standing endocarditis. In these cases it would have been impossible for the valves to close efficiently, and regurgitation must have taken place. The results of my observations, as I will show, demonstrate that tricuspid regurgitation must have been a factor in producing the wave *v* in the auricular venous pulse in these cases.

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[4] The Safety-valve Function in the Right Ventricle of the Human Heart, Guy's Hospital Reports, 1837, ii, 132.

[5] AMER. JOUR. MED. SCI., 1906, cxxxii, 393.

METHODS OF RECORDING THE MOVEMENTS OF THE CIRCULATION. The examination is best carried out with the patient lying down, with the shoulders slightly raised, and the head comfortably supported by a pillow, so that the sternomastoid muscle can easily be relaxed. A great variety of movements may be observed in different patients.

There are several ways of taking tracings of the movements in the jugular vein. Usually the movements are recorded on the blackened paper of a revolving drum or of a sphygmograph at the same time as some other standard movement, such as the apex beat or arterial pulse. These methods, besides being troublesome, permit only of tracings being taken for a short period, and it is often necessary in the study of irregularities to take tracings extending over a considerable time. I have lately devised a method whereby the lever of the instrument writes, with ink, on paper that is unwound from a roll, so that tracings many yards in length can be taken with much less trouble. For taking movements of the jugular and the carotid pulses or of the apex beat I employ a shallow cup (receiver). When this is applied over the pulsating part in such a manner that all communication with the outer air is prevented, the movement of the air within the receiver is conveyed by an India-rubber tube to the tambour. I have devised a special arrangement for taking a tracing of the radial pulse: the knob on the membrane of a broad tambour rests on a steel spring which is moved by the radial pulse, the steel spring being in a splint which is strapped to the wrist. A time marker, driven by a separate clockwork, records one-fifth of a second. Most of the tracings for this paper have been taken by this method.[9]

For the due appreciation of the movements in the jugular vein it is absolutely necessary that, along with the jugular pulse, the movements of some event whose position in the cardiac cycle is known, be taken at the same time as a standard, by which to determine the position in the cardiac cycle of any movement in the jugular pulse. For this purpose the arterial pulse usually offers the most convenient movement. Its cause—ventricular systole—is absolutely

[9] This instrument is made by Mr. S. Shaw, watchmaker, Padiham, Lancashire, England

determined. Fig. 1 shows the movements which serve as standards for the interpretation of a jugular pulse. I have placed below the jugular pulse, movements representing the apex beat and the carotid and radial pulses to show the relation in time of certain events in these various movements. The numbered down strokes indicate the time relations of the events in the jugular pulse, the apex beat, the carotid and the radial pulses: (1) The beginning of auricular systole; (2) the beginning of ventricular systole; (3) the opening of the semilunar valves and the appearance of the carotid pulse; (4) the beginning of the radial pulse; (5) the closing of the semilunar valves; and (6) the opening of the tricuspid valves. The space *E* between 3 and 5 indicates the time when the semilunar valves are open. These numbered down strokes and the space *E* have the same significance in all the other tracings. The time marked here and in all the other tracings equals one-fifth of a second.

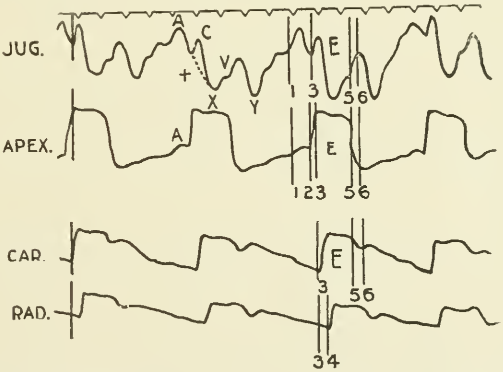

Fig. 1.—Tracings of the radial, carotid, and jugular pulses and of the apex beat.

Although the carotid pulse and the apex beat can sometimes be usefully employed as a standard, it will be found that the radial pulse is, on the whole, the most convenient. A certain loss of time takes place in the transmission of the wave to the radial pulse, and this can be estimated by taking simultaneously with the radial a few beats of the carotid, as in Figs. 2 and 10, in which the space between 3 and 4 shows the loss of time between the appearance of the carotid and the radial pulses. This loss being allowed for, one can always find a definite period in a jugular tracing which corresponds to any event occurring in the neck due to the ventricular systole. I also employ a very useful period, namely, that portion of the ventricular systole during which the semilunar valves are open (period marked *E* in all the tracings). Its duration can be found in the radial tracing, extending from the beginning of the up stroke to near the bottom of the dicrotic notch. It corresponds to the time between the down strokes 3 and 5 in all the carotid, jugular, and apex tracings given here. In the neck the period *E* begins with the carotid pulse.

There is a slight delay between the opening of the aortic valves and the carotid pulse, but it is so short ($\frac{1}{50}$ of a second) that it may be ignored. The space *E* in the radial tracing is about one-tenth of a second behind the same period in the neck.

Another standard which I will be able to point out later is that of the opening of the tricuspid valves (downstroke 6 in all the tracings), which in jugular tracings is always indicated by the beginning of the fall of the wave *v*. In the apex tracing this event occurs at the bottom of the fall after the systolic plateau as in Figs. 22 and 23.

INTERPRETATION OF A JUGULAR TRACING. 1. *The Auricular Wave* (*a*). To interpret such a tracing as Fig. 2 we find the space *E* (that is the period during which the semilunar valves are open). There is a wave *a* immediately preceding this period. From its position in the cardiac cycle this wave can only have been due to the auricular systole. Although it is recognized as being due to the systole of the auricle, there is some doubt as to its manner of production.

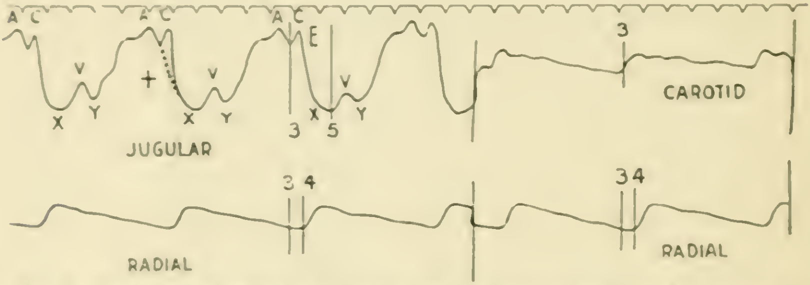

FIG. 2. Simultaneous tracings of the jugular and the radial pulses in the first part of the tracing and of the carotid and the radial in the latter part. The jugular pulse is of the auricular type. *a*, auricular wave; *c*, the carotid wave; *v*, the ventricular wave; *x*, the auricular depression; *y*, the ventricular depression. These letters have the same significance in all the other tracings.

Thus, it has been said to be due: (1) To a damming of the blood in the vein while the auricle is in systole; (2) to the fact that when an on-flowing stream is suddenly stopped there immediately takes place a wave in a contrary direction; and (3) to a true regurgitant wave. This is really a matter of some moment, as a due appreciation of this wave helps materially in understanding certain movements of the heart. Keith, as I have remarked, has described that regurgitation is prevented by the closure of the superior vena cava by the muscular band, the tænia terminalis. If the tonicity of this bundle be affected it will lengthen and become less effective. I am disposed to consider that it becomes readily affected by distention of the auricle under certain circumstances, even if it is ever really competent. In some of my cases in which there was a marked venous pulse, Keith has found this bundle greatly lengthened and atrophied, and I would therefore suggest that this wave is a true regurgitation wave and that it is due to inefficiency of the contraction of the tænia terminalis. I may point out in support of this view that Chauveau and Faivre[7] found in the horse that normally there is a slight regurgitation during auricular systole, which becomes markedly increased when the breathing is stopped.

2. *The Auricular Depression* (x). After the auricular systole there is a fall (x in all the tracings). It is usually the greatest depression in a tracing of the auricular form of venous pulse. If we leave out, for the moment, the consideration of the wave c, and consider the fall as following the dotted line at + in Figs. 1, 2, and 22, this fall will be found to occupy the whole period of E in many of the tracings, while in others it stops at varying periods before the end of E (Figs. 10, 12, 14, etc.). This fall is due to three factors, of which the first and principal is the relaxation of the auricle after its contraction, whereby the blood in the vein is suddenly passed into the dilating auricle. As a rule, it cannot be said how much is due to this factor and how much to the other factors, but in exceptional instances, in which we get a delay in the appearance of the ventricular systole, we find this fall divided in two, as (x and x^1) in Fig. 13.

After the auricular systole in this tracing, there is a slight delay before the wave c appears, and the fall x, between a and c, is due to the auricular diastole. The period E marks the time of ventricular systole, and we find, during this period, that there is another fall x.[1] The cause of x^1 is, as was first pointed out by John Hunter, the diminution of the intrathoracic pressure, caused by the expulsion out of the chest of the blood by ventricular systole. It is this diminished intrathoracic pressure, which causes the aspiration of the air in the respiratory tract, which has been studied by Mosso, Landois, François-Franck, Delépine, and others. There is no doubt that it must also have some effect on the flow of blood in the veins and in producing the fall x^1 in Fig. 13.

Keith has pointed out to me that another factor would be the enlargement of the auricular cavity by the dragging down of the auricular-ventricular septum during ventricular systole. Chauveau had previously described a similar action, and probably it is a factor in the production of this fall. In a tracing from a case of heart block Wenckebach[8] has also demonstrated a division of this fall and gives an explanation similar to this.

3. *The Carotid Wave* (c). The carotid wave c gave rise to much discussion in the earlier days, but I was able to demonstrate, in 1893, that it is really due to the impact of the carotid on the air in the receiver, and is not due to a wave in the vein. I was able to produce such ample evidence that this interpretation was generally

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[7] Nouvelles recherches expérimentales sur les mouvements et les bruits normaux du cœur. Gazette médicale de Paris, 1856, v, 406.

[8] Beiträge zur Kentniss der menschlichen Herztätigheit, Archiv f. Anatomie und Physiologie, 1906, 297.

accepted, but I find that Hirschfelder[9] and Morrow[10] have recently revived the old notion that it is in some way due to a wave sent back by the ventricle into the jugular vein.

It can be proved in many ways to be due to the carotid. Thus its synchronism with the carotid pulse is seen in Fig. 3. Clinically, we can demonstrate the nature of this wave by taking tracings from the neck at various levels. As we ascend higher in the neck the jugular element in the tracing will gradually disappear, while the carotid element will increase until nothing but the characteristic carotid pulse is left in the tracing. I have demonstrated this fact in many other ways, as, for instance, its absence when the jugular wave is large, as in Fig. 8, and its absence from the auricular liver pulse, as in Fig. 9. Morrow reckons to have demonstrated experimentally that this wave is due to the ventricular systole, but I am disposed to think that his experiments are not reliable. At all events, when able experimenters like Cushny and Hering took pure tracings of the jugular pulse they were able to get a tracing without a carotid wave.

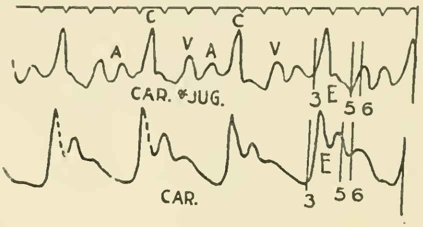

FIG. 3.—Simultaneous tracing of the jugular and the carotid pulses to show the synchronism of the wave *c* in the jugular with the carotid pulse.

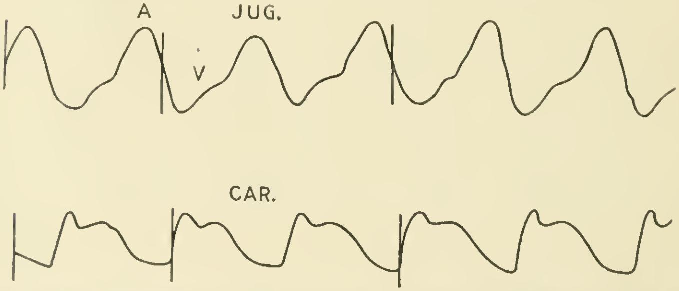

FIG. 4.—Simultaneous tracings of the jugular and the carotid pulses from a dog when the jugular had been dissected away from the carotid, showing the absence of a carotid wave in the jugular tracing (Cushny and Grosh).

Fig. 4 shows a jugular tracing from a dog taken by Cushny and Grosh after they had dissected the jugular vein away from the carotid.

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[9] Graphic Methods in the Study of Cardiac Disease, Amer. Jour. Med. Sci., 1906, cxxxii, 378.

[10] Discussion on Heart Block, Brit. Med. Jour., October 27, 1906, and The Various Forms of the Negative or Physiological Venous Pulse, Brit. Med. Jour., December 22, 1906.

It will be seen that there is no wave in the jugular tracing at the time of the ventricular systole, although in tracings taken prior to this dissection the carotid wave was always present. Likewise, Hering's[11] tracings, after he had detached the vein from the carotid, showed no sign of a wave at this time.

4. *The Ventricular Wave* (*v*). The next event to be considered is the wave marked *v* in all these tracings. It will be observed that I mark with *v* the small wave occupying a very short period in the cardiac cycle, in Figs. 1 and 22, as well as the large wave in Fig. 23, for the reason that the smaller wave is but the beginning of the larger. The best way to study this wave is to begin at its end. What causes the fall that terminates it? If careful measurements be taken to find in what portion of the cardiac cycle the fall occurs, the beginning of the fall will be found to occur at the time of the opening of the tricuspid valves (6 in all the figures). As a matter of fact, the fall of this wave is due to the opening of the tricuspid valves, permitting the blood that has been stored in the auricle during ventricular systole to flow into the ventricle, thus emptying the jugular vein. If this fact be kept carefully in mind, the real significance of the wave *v* becomes clear; its cessation marks a very definite period in the cardiac cycle and forms a valuable standard in the interpretation of many obscure tracings. The manner of origin of this wave is as follows: While the pressure in the ventricle during ventricular systole is higher than that in the auricle, the tricuspid valves will remain closed. The flow of blood into the auricle will continue to accumulate until the auricle is filled and the overplus will be stored in the venæ cavæ and adjacent veins. When the overplus is sufficiently great it will appear in the jugular as a wave. If the overplus is slight the capacious vena cava may retain it all, so that it will not appear in the jugular before the tricuspid valves are opened. The greater the overfilling of the auricle and the veins the earlier will be the appearance of the wave in the period *E* of ventricular systole.

The question is, Whence comes the blood that produces this filling and overfilling of the auricle? In the majority of people the jugular pulse is only apparent when the individual is recumbent. This posture favors the flow to the heart from the abdominal veins, and in certain circumstances this seems readily to lead to overfilling of the right heart. Another source to be considered is the regurgitation back from the right ventricle. I have examined very carefully the state of the heart in several thousands of patients who had a jugular pulse, and I have found that in many of them I could detect a tricuspid systolic murmur and slight dilatation of the right heart. If regurgitation does take place, in what way will it affect the jugular pulse? If the amount be slight it will only cause an earlier filling of the auricle, and this may be only sufficient to cause the overfilling to extend as far as the jugular, causing a wave to appear just before the opening of the tricuspid valves between the down strokes 5 and 6, as in Figs. 5 and 22. If, on the other hand, the regurgitation be more free the filling of the auricle will occur sooner and the wave appear earlier in the jugular tracing. This is, as a matter of fact, what I have found to take place.

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[11] Die Verzeichnung des Venenpulses am isolierten, kunstlich durchströmten Säugethierherzen, Archiv f. die ges. Physiologie, 1904, cvi.

Fig. 5 is from a man suffering from shortness of breath and tightness across the chest, in consequence of a very laborious occupation. There was slight enlargement of the heart and a well-marked tricuspid systolic murmur. Fig. 6 is from a profoundly anemic woman, with a large heart and a loud tricuspid systolic murmur, in whom there was a large pulsation in the veins of the neck. I have followed patients similar to these until they died, and found postmortem evidences of great dilatation of the tricuspid orifice.

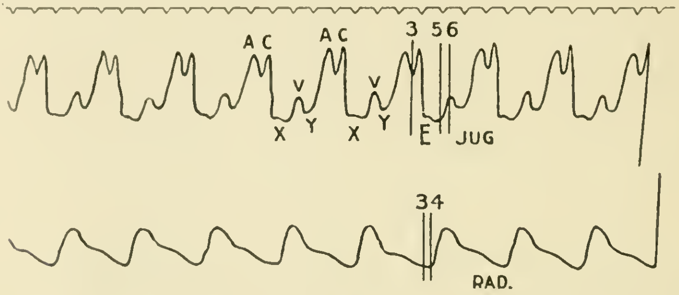

Fig. 5. Simultaneous tracings of the jugular and the radial pulses from a man with tricuspid regurgitation. The jugular pulse is of the auricular type and shows a small wave after the period *E*.

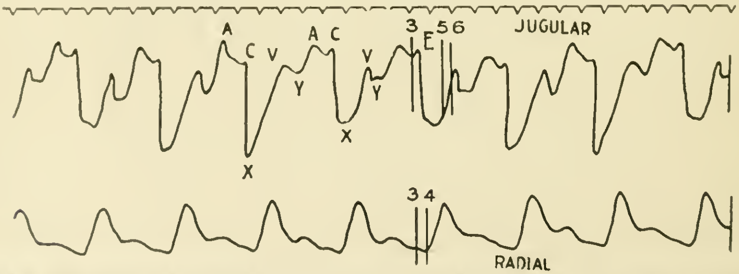

Fig. 6. Simultaneous tracings of the jugular and the radial pulses. The jugular pulse is of the auricular type. From a profoundly anemic woman with marked tricuspid regurgitation.

Thus, Fig. 7 is from a man who suffered from pernicious anemia. There was slight enlargement of the heart and a tricuspid systolic murmur. He died, and on postmortem examination the tricuspid orifice readily permitted the passage of four fingers. In such cases I suggest that the depressed tonicity of the muscular ring supporting the tricuspid valves permitted enlargement of the orifice and consequent incompetence of the tricuspid valves.

As some doubt may even here exist in regard to the diagnosis, I would call attention to instances of undoubted tricuspid regurgitation in patients in whom the tricuspid valve had been partially destroyed as a result of old endocarditis, and in which there could be no doubt of the fact that tricuspid regurgitation had occurred during life. I have had postmortem examinations on 8 cases of moderate tricuspid stenosis in which the opening permitted the passage of one or two fingers; in 6 of these there was pulsation of the auricular form in the jugular vein and the liver, and I had taken a large number of tracings of these pulsations.

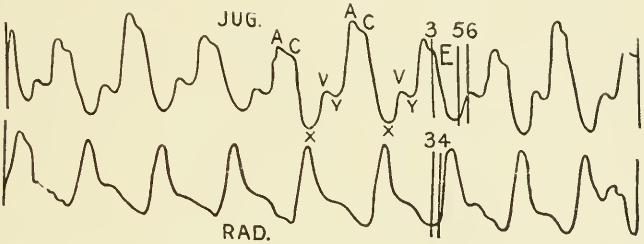

Fig. 7.—Simultaneous tracings of the jugular and the radial pulses from a man suffering from pernicious anemia. At the postmortem examination the tricuspid orifice was widely dilated. The jugular pulse is of the auricular type.

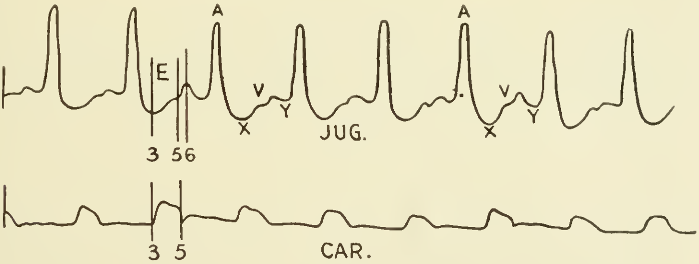

Fig. 8.—Simultaneous tracing of the jugular and the carotid pulses from a patient in whom the tricuspid valves were found damaged by an old endocarditic process, and the tricuspid orifice thereby rendered markedly incompetent. The jugular pulse is of the auricular type.

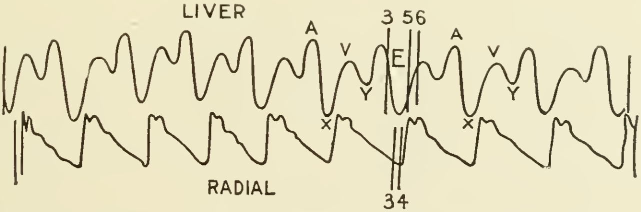

Fig. 9.—Simultaneous tracing of the liver pulse and the radial pulse from another patient whose tricuspid valve had been rendered incompetent because of an old endocarditis. The liver pulse is of the auricular type.

Fig. 8 is a tracing of the jugular pulse from one case, and Fig. 9

is a tracing of the liver pulse from another. In an analysis of these tracings it will be found that the wave *v* is small and appears only toward the end of ventricular systole, and its fall begins at the time of the opening of the tricuspid valves. That is to say, in the people from whom these tracings were taken, tricuspid regurgitation undoubtedly occurred, and in doing so it must have modified the wave *v* in the tracings. I cannot conceive how it could affect these tracings in any way other than in participating in the earlier filling of the auricles and veins and, therefore, in the production of the wave *v*.

I have selected purposely, to illustrate my argument, cases in which there could be no reasonable doubt as to the occurrence of tricuspid regurgitation, in order to impress the fact that this factor must be concerned in the production of the wave *v*. As I have said, I have taken tracings of several thousand cases and have studied at the same time the condition of the heart. In the large majority of cases no tricuspid murmur could be heard, and in many cases there was practically no increase in the size of the heart.

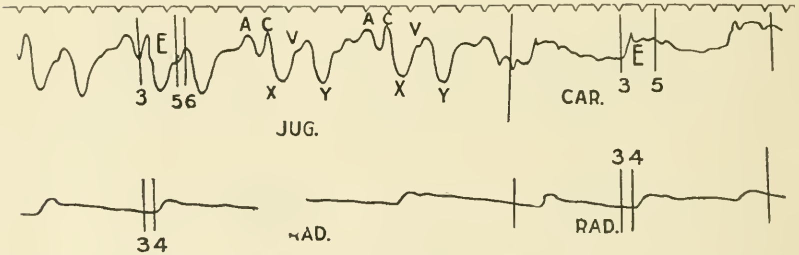

Fig. 10.—Simultaneous tracing of the jugular and the radial pulses in the first part of the tracing, and of the carotid and the radial pulses in the latter part. From a man without a tricuspid systolic murmur.

Thus, Fig. 10 was taken from a pale, spare man, aged twenty-five years, with dilated stomach. There was no cardiac enlargement and no systolic murmur. He was rather short-winded. The wave *v* encroaches on the period *E*. What is the explanation of such cases? Tricuspid regurgitation is not excluded by the absence of a systolic murmur. One can frequently demonstrate the existence of large tricuspid regurgitation, as shown by a ventricular venous pulse, with the absence of a tricuspid regurgitant murmur. Careful clinicians are well aware that under certain circumstances regurgitation can occur at all the orifices with no murmur. Thus, for instance, in the enormously dilated hearts of adherent mediastinitis there may be no systolic murmur though the mitral and tricuspid orifices are greatly widened. The question, therefore, whether tricuspid regurgitation takes place in such cases as the man from whom Fig. 10 was taken is one on which I have not sufficient evidence to decide. As I have already pointed out, the essential element in the production of the wave *v* is a sufficient accumulation of blood in the right auricle, so that the auricle and superior vena cava are overfilled during the period of the cardiac cycle when the tricuspid valves are closed. This can occur with a slight engorgement of the right heart, when there is an increase in the residual blood after the auricular systole. The jugular pulse in these cases readily disappears with more rapid action of the heart, as on moderate exertion, or on the supervention of a slight febrile attack, and often increases in size with a slower action of the heart. It is probable the mere increase of the accumulated blood in the right heart may suffice alone to produce the wave v in such cases, but whether it may at the same time render the tricuspid valve incompetent is a question that must be considered, but which I am unable to answer.

Before demonstrating how the auricular venous pulse passes into the ventricular, I will consider some of the objections raised against the foregoing explanation. The erroneous notion very commonly raised by people who have not fully considered all the factors concerned and found in text-books, physiological and clinical, is that if there were tricuspid regurgitation in such cases as in the foregoing instances the wave v ought to appear at the beginning of ventricular systole and not at the end. If the jugular vein was in such direct communication with the right ventricle as the carotid is with the left, then undoubtedly the wave v should appear at the same time as the carotid. *But it must be borne in mind that between the right ventricle and the jugular vein there is interposed a dilating auricle and a capacious superior vena cava, and that a wave can only appear in the jugular after the auricle and vena cava have been overfilled.* It is for this reason that the time of the appearance of the wave v in the period E is a measure of the engorgement of the right auricle, as I shall demonstrate shortly.

It has been suggested by Gerhardt, Wenckebach, and others that the wave v in such tracings as Figs. 2 and 5 is due to a backward movement of the auriculo-ventricular septum after the closure of the semilunar valves. They state that at this period, when relaxation of the ventricle takes place, the septum swings back, sending a wave through the auricle and superior vena cava into the jugular. I am too ignorant of the movements of the heart to say whether there is such a movement at this time, and the arguments given in favor of it are more presumptive than demonstrative. Admitting, however, that it does occur, it would only be a factor added to those I have already given, and would not account for the portion of the wave occurring during the period E in such tracings as Fig. 10. I called attention, in 1893, to the fact that there is often a notch in the jugular tracing at the time of the closure of the semilunar valves, but its cause is still obscure. If this return of the auriculoventricular septum does take place it cannot be of such great force, as, for instance, to distend the liver and raise one's hand when laid on the liver, for one can see the hand raised by this wave, in palpating the liver in cases of auricular pulsation of that organ. Another curious feature is that those who support this view leave out of consideration altogether the manner in which this wave *v* grows forward into the period *E*. Lately two physiologists, Hering and Morrow, by experiments on dogs, have sought to show that this wave is a normal sign and has nothing to do with tricuspid regurgitation. Their experiments are not conclusive, for they do not seem to have tested whether the tricuspid valves were competent, nor did they seek to produce tricuspid incompetence and observe the result on such incompetence. I do not see how they can say that tricuspid incompetence did not participate in the production of the waves in the cases cited here when the tricuspid valves were damaged.

5. *The Ventricular Depression* (*y*). The next event after the wave *v* to be seen in the jugular and liver pulsation is the fall marked *y* in all these tracings. Its cause is readily understood, being due to the passage into the ventricle of the blood stored in the auricle during ventricular systole. It will be noticed that in all these tracings the fall begins at the down stroke 6, that is at the time of the opening of the tricuspid valves. If the heart be slow in rate it is followed by a rise shown in Fig. 2, between *y* and *a*. This rise is due to stasis in the veins. If the heart quickens in its action the period of stasis first disappears and the fall *y* is followed at once by the auricular wave. If further quickening occurs the fall *y* disappears from the tracing, and the wave *v* becomes continuous with the auricular wave, as in Fig. 11.

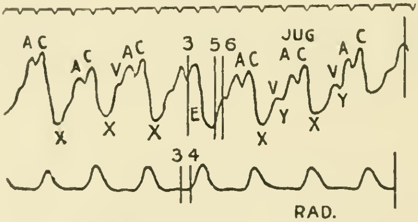

FIG. 11.—To show the disappearance of the fall *y* and the wave *v* with quickening of the heart rate.

In my book on the pulse I have given numerous illustrations of these features, so that I need not dwell on them here. As a rule, the fall *x* is more marked than the fall *y*, in the auricular form of the venous pulse; but, as the pulsation passes into the ventricular form the fall *y* gradually becomes the more pronounced, and the fall *x* diminishes, until it disappears altogether, until there is but one large wave synchronous with and due to the ventricular systole, and one great fall *y*, synchronous with and due to the ventricular diastole. The manner in which this occurs I will now proceed to demonstrate.

THE PASSAGE OF THE AURICULAR FORM OF THE VENOUS PULSE INTO THE VENTRICULAR. The reason I called the wave marked *v* the *ventricular wave* in the foregoing tracings, although it may be that the ventricular systole in many instances takes no part in its production by sending blood back into the auricle, was to impress the fact that had been overlooked, that tricuspid regurgitation must have had a beginning, and that the "positive" or ventricular jugular pulse, which everyone recognizes as due to extreme tricuspid regurgitation, began its appearance as a small wave appearing at the end of ventricular systole. How necessary it was to insist on this fact is seen in the writings of those who have not recognized early tricuspid regurgitation. Scarcely a writer ever attempts to explain how the "positive" venous pulse comes about. The reason for this silence is, that these writers, clinicians as well as physiologists, have failed to recognize the important factor of slight and moderate tricuspid regurgitation. I quite admit that to follow my argument it may require a clearer exposition than I am capable of giving, but my facts cannot be gainsaid. If, therefore, those interested in this matter will ponder deeply on this somewhat complex problem, I am convinced they will see the explanation as clearly as I do.

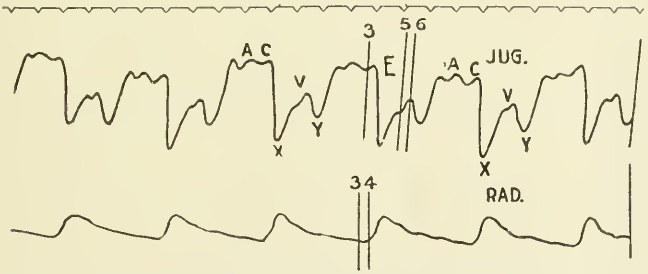

Fig. 12.—Shows the extension forward of the wave v into the period E when the patient held his breath. Compare with Fig. 5, taken from the same patient.

While the following explanation shows how the auricular form of the venous pulse passes into the ventricular, it affords, at the same time, proof of varying degrees of engorgement of the right heart and demonstrates the manner in which an increasing engorgement can be recognized in tracings of the jugular pulse. I am somewhat at a loss in selecting illustrations, as no two cases are alike in all particulars, and I have by me such an abundance of material illustrating the view I am propounding, that in the limits of this paper I am restricted to giving only a very small portion of my proofs. In suitable cases it is possible to increase the amount of right-heart engorgement by getting the patient to hold his breath. In many instances in which the jugular pulse is of the ventricular type, it may occasionally disappear, but can be brought back by holding the breath. In some cases in which the jugular pulse is of the auricular form the wave v can be made to increase in size and to appear earlier in the cardiac cycle by the same method. Thus, Fig. 5 shows v appearing only after the period E. When I told this patient to hold his breath for a long time the jugular pulse increased in size and the wave *v* extended forward into the period *E*, as in Fig. 12.

Here I reason that the greater resistance offered to the systole of the right ventricle by the cessation of the breathing had caused a rise in the ventricular pressure and an increased backward flow. This explanation corresponds with that of Hering's[12] experiment when he found he had difficulty in raising the pressure in the right ventricle, because of the ease with which the tricuspid leaked.

The best evidence of increasing engorgement is to be found by watching individual cases for many years and noting the gradual advance of the cardiac inefficiency. The following case is a good illustration of this method: I attended a man during an attack of acute endocarditis, in 1881, he then being thirty-two years of age. He was left with a mitral regurgitant murmur. In my notes of his case I find a tracing of his jugular pulse taken in 1892 (Fig. 13), which shows that the wave *v* began a little before the end of the period *E*. At that time he was in fair health. It will be noted in the tracings from this patient that the carotid wave *c* follows the wave *a* at a greater interval than in the other tracings of the jugular pulse given in this paper. This delay implies a depression of conductivity of the fibers joining auricle and ventricle. In some tracings,

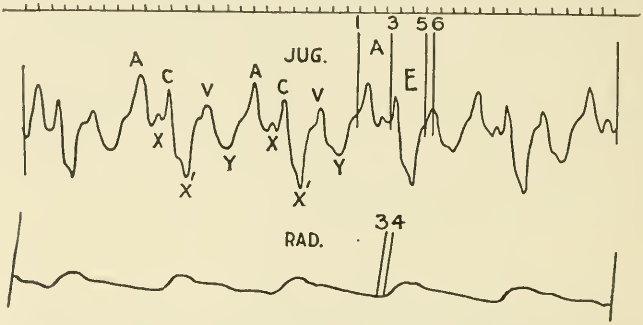

FIG. 13.—Note the size of the wave *v* in this and the following tracings. W. H., 1892.

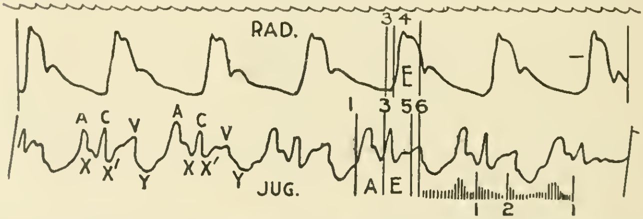

FIG. 14.—The shading underneath the jugular tracing shows the position of the heart murmurs. 1 and 2 refer to the first and second sounds. Note the increase of the diastolic murmur at the time of the auricular systole. W. H., 1903.

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[12] Zur experimentellen Analyse der Unregelmassigkeiten des Herzschlages. Archiv f. die ges. Physiologie, Bonn, 1900, lxxxii.

taken in 1897, I find this had increased so much that there was a slight heart block. In 1898 he suffered from an attack of grievous heart failure, from which his recovery was slow and protracted. In 1903, I took the tracing, Fig. 14. Here it will be noted that the wave *v* appears still earlier in the period *E*. At this time there was, in addition to the mitral systolic murmur, a long diastolic murmur with a curious increase of intensity a little before the first sound, a brief interval occurring between this increase and the first sound, corresponding to the shading underneath the jugular tracing. It will be seen that the rise in the shading occurs exactly at the time of the auricular systole. It was my custom to ask my colleagues when showing them this patient, to draw for me on a tracing the period in the cardiac cycle in which this increase in the intensity of the diastolic murmur occurred, and in every instance they placed the period of increase exactly where I have shown it, although they could form at the time no opinion as to the cause of the increase. There can be no doubt that the increase in the loudness of the murmur at this time was due to the auricular systole.

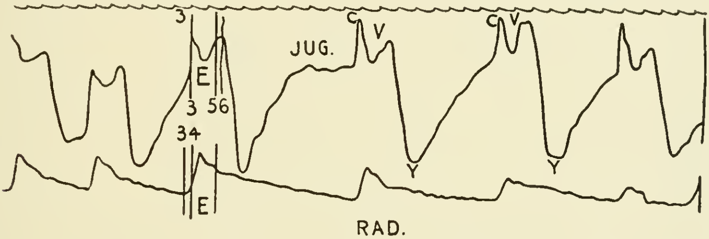


FIG. 15.—Simultaneous tracing of the jugular and the radial pulses. Note the irregularity and the absence of an auricular wave as compared with Figs. 13 and 14. The jugular pulse is now of the ventricular type. W. H., April 6, 1904.

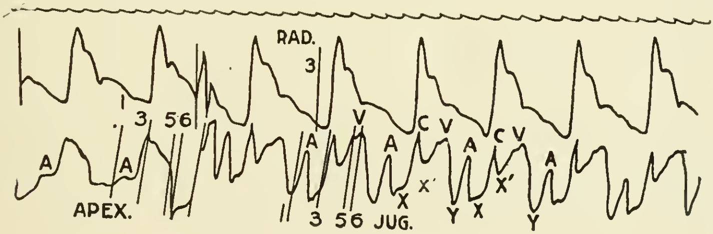


FIG. 16.—Simultaneous tracings of the radial pulse and the apex beat in the first part of the tracing and of the radial and the jugular pulses in the latter part. The pulse is again regular and the auricular wave *a* has returned to the apex beat and the jugular pulse. W. H., April 11, 1904.

The character of the jugular pulse on repeated examinations continued like Fig. 14 until April 6, 1904, when I found the pulse continuously irregular, and on listening to the heart there was a systolic and a diastolic murmur at the apex, but no increase in the diastolic murmur before the first sound. On taking a tracing all sign of the auricular wave had gone from the jugular pulse and there was but one large wave during the ventricular systole (Fig. 15). A week later the pulse was found to be quite regular and the jugular tracing showed the auricular wave had returned (Fig. 16). At the beginning of this tracing there are a few beats from the apex which show a wave, due to the auricle. I have taken a large number of apex tracings and never failed to find this wave when the rhythm was regular. The heart continued quite regular until the month of August, 1904, when I found it again continuously irregular and it has remained thus ever since.

Figs. 17 and 18 were taken on December 9, 1906, and are typical of the form of arrhythmia that I have found in the numerous tracings I have taken during the last two years—one large wave synchronous

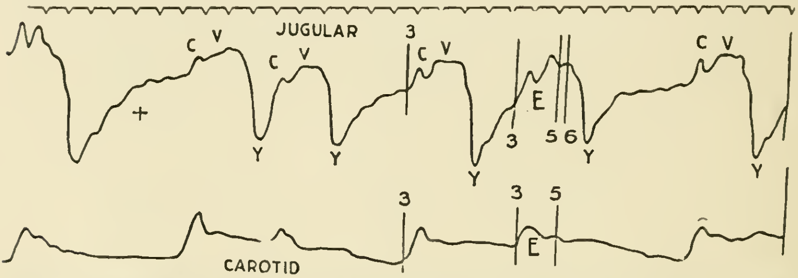


FIG. 17. Simultaneous tracings of the jugular and the carotid pulses. The jugular pulse is now of the ventricular type. Note the irregularity and the small ripples in the tracing at the long pause +, due in all likelihood to fibrillary contraction of the auricle.

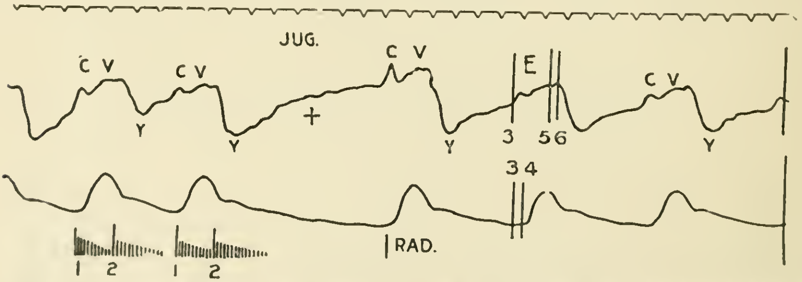
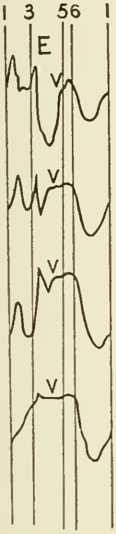


FIG. 18. Here the jugular pulse is taken with the radial pulse and shows the same features as Fig. 17. Note the shading showing the murmurs and sounds of the heart, and compare with Fig. 14. W. H., 1906.

with the ventricular systole, no sign of an auricular wave in the tracing of the apex beat or the jugular pulse, no murmur due to the auricular systole, and invariably irregular. To appreciate more clearly the changes in the jugular pulse I arrange one cardiac cycle from Figs. 13, 14, 16, and 17, above one another, showing thus in a semi-diagrammatic form the gradual increase of the wave *v*, and bringing out clearly its meaning (Fig. 19). This demonstration of the passage of the auricular form of the venous pulse into the ventricular justifies the names I have given them, for it is seen in these tracings that the disappearance of the auricular wave marks at once a remarkable change not only in the character of the venous pulse but also in the rhythm of the heart. I wish to insist upon these facts, because though much has been written on mitral stenosis the clinical signs have never been properly appreciated, not even the fact that when the heart becomes continuously irregular, as it often does in advanced mitral stenosis, a presystolic murmur of the crescendo type



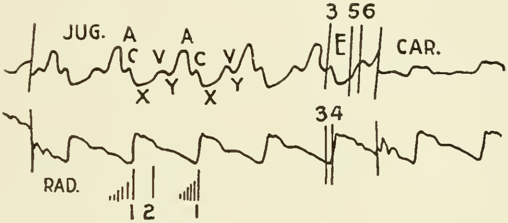


Fig. 19.—Semidiagrammatic illustration of the passage of the auricular form of the venous pulse into the ventricular. Note the gradual increase in the size of the wave *v*.

Fig. 20 is from a case of mitral stenosis, taken in 1896, and shows a regular heart and a jugular pulse of the auricular form. The crescendo murmur is represented in the shading running up to the first sound. Mrs. P., 1896.

Fig. 19

Fig. 20

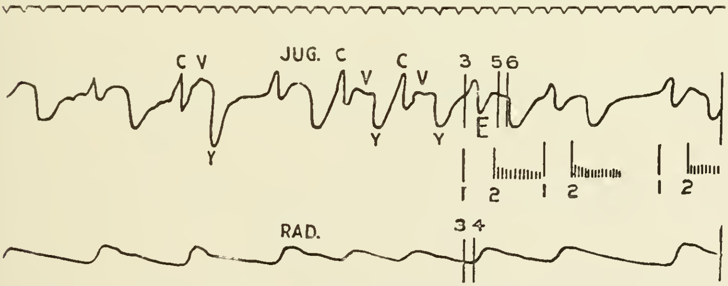


Fig. 21.—From the same patient as gave Fig. 20, taken in November, 1906. The rhythm is now irregular and the jugular pulse is of the ventricular type. The shading shows a diastolic murmur and an absence of the crescendo character represented in Fig. 20. Mrs. P., 1906.

is never heard. A diastolic murmur may be present and it may last during the whole of a short diastole, but if care be taken it will be found never to terminate in a crescendo murmur, and when the diastole is prolonged the murmur will be found to stop short of the first sound, as shown in the shading of Fig. 21.

Fig. 20 is a tracing taken in 1896, from a woman with mitral stenosis. The pulse is regular and the jugular shows the usual form of auricular venous pulse. The shading shows the position of the crescendo presystolic murmur running up to the first sound. In 1900 her heart became irregular and has remained so ever since. Fig. 21 is a tracing taken in 1906, and is characteristic of the irregularity and type of venous pulse—the ventricular—that has been present the past six years.

When the patient's heart beats quickly, as after exertion or excitement, the rate being rapid, the rhythm is fairly regular and a diastolic murmur fills up the whole of the diastolic period and might be mistaken for a presystolic murmur. But there is no crescendo character present in it. When, however, the heart becomes quieter, the irregularity becomes more marked, and long diastolic pauses ensue, and then it will be found that the diastolic murmur does not fill up the whole diastolic interval but stops short, as shown in the shading of Fig. 21. I have been able to demonstrate this peculiarity in a great number of cases to many of my colleagues. The study of the jugular tracings at once reveals the reasons for these changes. Progressive narrowing gives rise to, first, a crescendo presystolic murmur, then there is added a diastolic murmur, then the auricle ceases to beat at its normal period of the cardiac cycle and the crescendo murmur disappears, and the heart becomes irregular. In advancing mitral stenosis, therefore, these events happen simultaneously: (1) Disappearance of the auricular wave from the jugular pulse and apex beat; (2) disappearance of the auricular systolic (crescendo presystolic) murmur; and (3) arrhythmia of the heart. So far as the venous pulse is concerned this means the change from the auricular form to the ventricular.

It so happened that my earlier cases of the ventricular form of the venous and the liver pulses were all secondary to disease of the mitral valve, and at the postmortem examinations I had found the right auricle enormously distended and the muscle fibers greatly atrophied, so that I surmised that the auricle, like an overdistended bladder, had become paralyzed. This view seemed to be confirmed by the results of careful observation of individual patients extending over many years, the auricle never putting in an appearance at its normal period in the hundreds of tracings I have taken. This view I expressed in my book, but when writing it a few cases came under my observation that gave me grounds for suspecting that "auricular paralysis" was not the only condition that gave rise to the ventricular form of the venous pulse.

I, therefore, started on an inquiry into all forms of arrhythmia, and collected a large number of cases. From among these I was able to differentiate a group of over 500 cases in which the irregularity was of a disorderly nature, as in Figs. 21 and 23. Over one-half of these cases had a venous pulse, and in every case it was of the ventricular form. In more than 50 cases the disorderly rhythm had started when the patient was under observation, and it invariably meant the change from the auricular form to the ventricular—often suddenly. Usually, these cases for years have an occasional irregularity due to ventricular extrasystoles, that is, when the ventricle contracts before the auricle and independently of the stimulus from the auricle. I shall cite only one case as an illustration, particularly, as it has some historical value, being the first human subject in whom I demonstrated the nature of ventricular extra-systole.

In 1892, I took the tracing Fig. 22. In many tracings taken from this patient up to 1906 the venous pulse was always of the auricular form, as in this tracing, though she frequently showed ventricular extrasystoles, which are given in my book (Figs. 69 and 279). I saw her on August 6, 1906. The pulse had suddenly become quick and irregular and the jugular pulse was of the ventricular type. By November it had slowed down, and Fig. 23 is a characteristic tracing of its present condition.

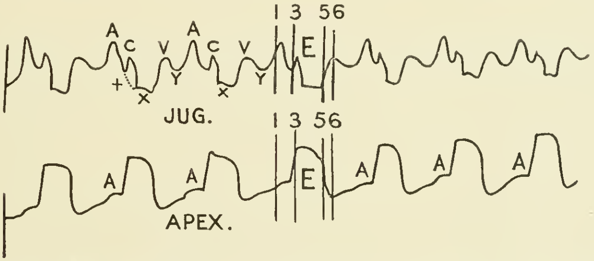


Fig. 22.—Simultaneous tracings of the jugular pulse and the apex beat. The jugular pulse is of the auricular type. Compare with Fig. 23. Mrs. A., 1892.

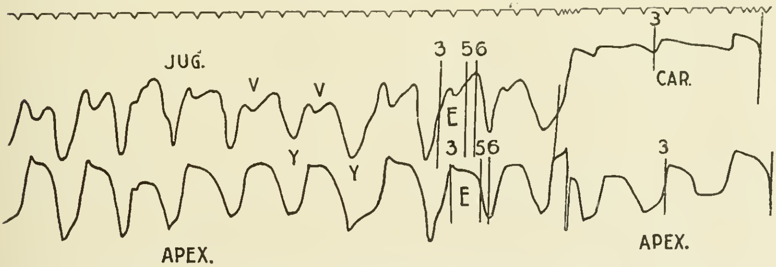


Fig. 23.—Simultaneous tracings of the jugular pulse and the apex beat. The jugular pulse is now of the ventricular type. Mrs. A., November, 1906.

Compare here the disorderly rhythm, the absence of the auricular wave in the jugular, and the apex tracing, with the perfect regularity and the presence of the auricular wave in the jugular and apex tracing of Fig. 22. Note, however, the persistence of the wave v between the down strokes 5 and 6 in both cases.

What is the auricle doing in cases with the ventricular venous pulse? I had assumed that it was paralyzed, and the total absence of any sign of its action for years, and the extreme distention of the chamber and its atrophied walls seemed to support the view. In some cases I have found evidence that it contracts at the same time as the ventricle, and on postmortem examination these cases did not show extreme atrophy of the auricular muscle. In a few other cases I sometimes found a series of small waves on the ascending line of the tracing preceding the ventricular wave, as at + in Figs. 17 and 18. The waves are sometimes larger than those in these tracings, as at + in Fig. 24, which was taken from the same patient that gave Fig. 22 and 23.

In discussing this matter with Professor Cushny he said he had no hesitation in accepting the view that these small movements are due to fibrillary contraction of the auricle, as he had frequently witnessed the auricle in fibrillary contraction while the ventricle went at a slower rate.

One conclusion I have come to, and it is this, that when the ventricular venous pulse is present the heart's contraction proceeds no longer from the normal place, that is, at the mouths of the great veins, but probably at the fibers joining the auricle and the ventricle. At present, Dr. Keith is conducting a minute inquiry into the condition of these fibers in a number of hearts I sent him of patients from whom, during life, I have taken numerous tracings, and so far it would seem that he will be able to show some anatomical facts justifying the foregoing conclusion. If this is so it will throw a flood of light on many obscure cases of heart failure which are at present buried under such vague terms as "chronic myocarditis," "fatty heart," "mitral irregularity," etc. It opens up a new view to clinicians and physiologists, and its bearing on treatment is of the very greatest importance. The heart failure is directly due to the arrhythmia, for the parts of the heart no longer work in that orderly manner designed by nature; consequently, treatment has a very definite aim before it. Further, the effect on these hearts of such a drug as digitalis is entirely different from the effect on a heart beating with a normal rhythm.

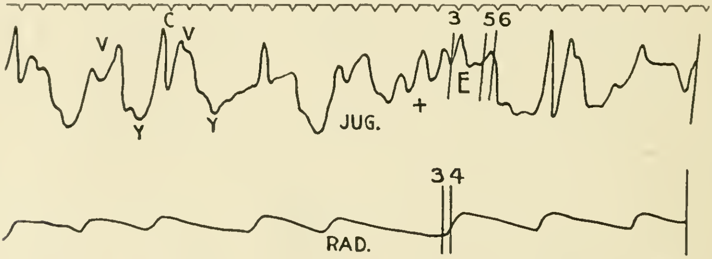


FIG. 24. The jugular pulse is of the ventricular type and shows three small waves at + probably due to fibrillary contraction of the auricle.

I hope that I have been able to show how necessary it is for the proper appreciation of the functional pathology of the heart, to understand the meaning of the venous pulse, and also to show that a great field for research still lies before the student of clinical medicine.

BACTERIAL INOCULATION IN THE TREATMENT OF LOCALIZED INFECTIONS.

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In June, 1906, the necessary equipment was installed in the Pathological Laboratory of the Montreal General Hospital for the prosecution of research and clinical work in the field of bacterial inoculation, after a first-hand experience by one of us in the laboratory of Professor A. E. Wright, at St. Mary's Hospital, London. Ample material was promptly placed at our disposal, and from a series of upward of fifty cases a selection based upon the following considerations has been made: (1) Cases yielding the most brilliant results from the point of view of the time required to effect a cure, and (2) cases representative of different infections or varying clinical types of the same infection.

Technique of the Method of Preparing Bacterial Vaccines. The method here described in detail applies to the preparation of vaccines from the staphylococcus group. For variations as to media, age of cultures, devitalizing temperature, dosage, etc., one should consult the appended table and special remarks.

In cases of acne and furunculosis material for cultural purposes is obtained by puncturing the lesion with a sterilized needle, after washing the cutaneous surface with alcohol and ether. The pus expressed is transferred by the aid of a platinum loop to a plain agar slant, care being taken to ensure an even distribution of the material over the entire surface. This may best be effected by employing several loopfuls of the water of condensation. In order to secure the highest efficiency it is desirable that the vaccines be prepared from the original cultures or from first transplants.

After incubation for from twelve to eighteen hours the growth, if in pure culture, is washed from the surface of the slant with 6 c.c. of 0.85 per cent. saline solution, and decanted into a sterile test tube. The tube is then drawn out and sealed in the blowpipe flame and the contents agitated for ten minutes to ensure an even emulsion. The tube is then reopened, a drop of the emulsion for standardizing purposes is expelled, and the tube is resealed and immersed in the water-bath for one hour at 60° C. Finally a control culture is made and placed in the thermostat for twenty-four hours, at the end of which time, if negative, the vaccine may be diluted for use.

STANDARDIZING. Into a long narrow pipette fitted with a rubber teat and upon which a volume has been marked with a blue pencil, equal volumes of finger blood and the emulsion withdrawn before devitalization are aspirated, together with one or more volumes of 0.85 per cent. saline solution as a diluent. The contents of the pipette are then expelled upon a slide, thoroughly mixed by repeated aspiration, and films drawn. The latter are then stained by Leishmann's or Wright's blood stain and 500 red cells counted, noting at the same time the number of cocci present in the different fields. By a comparison of the two the number of cocci per cubic millimeter may be readily determined. Thus if in counting 500 red cells 500 cocci are encountered the vaccine contains 5,000,000 cocci per cubic millimeter or 5,000,000,000 per cubic centimeter.

DILUTING AND LYSOLIZING. For diluted vaccine 50 c.c. amber-glass bottles are employed. These bottles are filled with 50 c.c. of 0.85 per cent. saline solution, plugged with cotton-wool, and autoclaved. To continue our example of a vaccine standardized at 5,000,000,000 per cubic centimeter: After determining the cubic contents of the vaccine tube (or comparing it with a tube of equal size filled to the same level with water and measured) 5 c.c. of the contents of the bottle is poured into a cubic centimeter graduate, and replaced by an equal quantity of the concentrated vaccine. We thus have an emulsion 1 c.c. of which contains 500,000,000 staphylococci. We next add 0.25 per cent. of pure lysol—that is 2.5 milligrams per cubic centimeter. Finally a sterilized rubber cap is drawn over the neck of the bottle, and the mouth of the bottle immersed to the shoulder in hot paraffin. In withdrawing a vaccine for use the hypodermic needle is plunged through the rubber cap after sterilizing the surface in the flame. The puncture is closed by the same procedure. In calculating dilutions it is desirable that the minimum dose should not be less than 0.25 c.c.

SPECIAL REMARKS. While in order to save time the use of a "stock" vaccine may be employed, that is a vaccine prepared from the infective organism in a similar clinical condition, a "personal" or autogenous vaccine should be prepared from the infecting agent in each individual case if the best results are to be obtained. Personal vaccines of staphylococcic infections are chiefly of importance in the treatment of acne, whereas in furunculosis and carbuncle, heterogenous vaccines not infrequently yield surprising results.

In the treatment of staphylococcic infections apart from acne it has been our practice to employ for the initial dose a vaccine which has previously yielded prompt results in the treatment of lesions presenting similar clinical features; thus a "personal" vaccine prepared from a case of furunculosis may be subsequently employed as "stock" vaccine in the treatment of a similar clinical condition. The same remarks apply to sycosis, carbuncle, and impetigo infections.

In sinus infections the preparation of "personal" vaccines is somewhat more complicated, as there are usually present in addition to Staphylococcus aureus and albus one or other of the large group of saprophytes, necessitating careful plating of the original culture. If Staphylococcus aureus and albus (or citreus) are present a mixed vaccine should be employed. The same laborious procedure is usually necessary in order to isolate the pneumococcus from the discharge of an empyemic cavity. In streptococcic infections the personal vaccine appears to be a *sine qua non.*

In Bacillus coli infections of the bladder there is seldom much difficulty in isolating the organism in pure culture, and in cases in which this organism alone is responsible for the symptoms, inoculation yields prompt results.

In meningococcal infections the value of the personal vaccine has not yet been demonstrated, but from our experience with gonococcal infections, if any analogy may be assumed, autogenous vaccines are, as a rule, the least effectual. So far we have employed but one strain of the meningococcus, with excellent results. In order to isolate this organism with reasonable certainty the cerebrospinal fluid obtained by lumbar puncture should be placed in the thermostat for twenty-four hours, then subjected to sedimentation in a high-speed centrifuge, and the residue transferred to blood-agar slants. By this means an abundant growth is usually obtained after twenty-four hours' incubation, even in cases in which numerous slides from the original fluid fail to show the presence of cocci. Vaccines should then be prepared from transplants, and incubated for from twelve to eighteen hours. The washings from several tubes may be employed if growths are scanty, but the use of young cultures is of paramount importance for accurate standardizing, as autolysis is usually early and rapid, so that vaccines prepared from old cultures may be of a strength out of all proportion to the numerical count.

In the preparation of gonococcal vaccines six-hour growths upon hydrocele-agar yield the best results. In older cultures it has been found impossible by simple agitation to obtain an even emulsion, in the absence of which standardization cannot be considered accurate owing to the presence of large clumps which do not permit of enumeration.

Tubercle Vaccine. In the treatment of localized tuberculous lesions we have heretofore employed Koch's T. R. tuberculin, which may be obtained in 1 c.c. containers, representing in suspension 10 milligrams of devitalized, dried, and ground tubercle bacilli. Dilution should be carried out in the following manner:

1. Immerse the container for ten minutes in pure lysol, wipe with a sterile towel, open, and decant into a standard vaccine bottle containing 39 c.c. of 0.85 per cent. saline solution, which has previously been autoclaved; rinse the container with 1 c.c. of fluid from the vaccine bottle; add to the whole 0.25 per cent. of pure lysol or 100 milligrams. Adjust the rubber cap and seal as before described. 1 c.c. of this

first dilution contains 0.25 milligram of tuberculin. This dilution is used as "stock."

2. After thoroughly shaking withdraw through the rubber cap by means of an accurately graduated syringe fitted with a hypodermic needle 1 c.c. of this "stock" dilution, and transfer it to a second vaccine bottle containing 49 c.c. of autoclaved saline solution, lysolize and seal as before. This dilution constitutes "Tuberculin No. 1," 1 c.c. of which contains $\frac{1}{200}$ milligram.

3. From this second dilution withdrawn by the same means 10 c.c. and add it to 30 c.c. of autoclaved saline solution contained in a third vaccine bottle. Lysolize by adding 75 milligrams of pure lysol. This third dilution constitutes "Tuberculin No. 2," 1 c.c. of which contains $\frac{1}{800}$ milligram of T. R. tuberculin.

It is of great importance that each step should be carried out with the strictest precautions against contamination and that each dilution should be carefully labelled so that the possibility of administering an overdose may be reduced to a minimum. The dose should be $\frac{1}{800}$ to $\frac{1}{2000}$ milligram according to body weight and the nature of the lesion. It is better practise to begin with a minimal dose, say $\frac{1}{1600}$ milligram, until the degree of reaction is known through subsequent determinations of the opsonic index, and when the negative phase is pronounced or prolonged or the patient complains of headache and general malaise it is well to adhere to the minimal dose for the first few inoculations. An exception to this rule of minimal primary dosage is, in our opinion, to be found in cases of infection of the urinary tract, in which the maximum dose, that is $\frac{1}{800}$ milligram, may be given at the first inoculation.

We have attempted the preparation of "personal" tuberculous vaccines employing the same general technique as for other organisms (see Case I) amplified as follows:

Cultures from bouillon flasks should be taken as soon as the growth has reached the margin of the fluid, that is before corrugation of the surface or precipitation has occurred. The cultures are first immersed in the water-bath for two hours at 60° C., in order to devitalize. The bouillon is next removed by siphon, and the residue washed in 0.85 per cent. saline solution, and centrifugated until the overlying fluid is perfectly clear. This fluid is then decanted and the mass of tubercle bacilli dislodged into a sterile agate mortar, where it is ground for one hour, sufficient saline being added from time to time to maintain a pappy consistence. This finally divided material is then diluted with 10 c.c. of saline solution, centrifugated for fifteen minutes to get rid of the clumps, and the supernatant emulsion is siphoned off into a sterile test tube. The residue is again ground, diluted, and centrifugated. This emulsion is added to that already obtained and the test tube is sealed and thoroughly agitated; after withdrawing a specimen for standardization, the vaccine is once more heated in the water-bath for two hours at 60° C. Finally this is diluted and lysolized. The minimal dose is 500,000.

While this method is open to certain objections, it is one which may be carried out by those in possession of a comparatively meagre laboratory equipment. Time alone will prove whether or not the actual disintegration of the organism by crushing is essential. By the method outlined, though the emulsion is free from clumps, the individual organisms for the most part remain intact.

Organism.	Media.	Age of culture: hours.	Devitalizing temp. one hour.	Dosage: millions.
Staphylococcus	Plain agar	12 to 18	60° C.	100 to 500
Streptococcus	Plain agar	24	58° C.	10 to 20
Pneumococcus	Serum	24	58° C.	50 to 100
Bacillus coli communis	Plain agar	8	54° C.	50 to 100
Bacillus pyocyaneus	Plain agar	12	54° C.	50 to 100
Gonococcus	Hydrocele-agar	6	60° C.	10 to 20
Meningococcus	Blood agar	18	60° C.	10 to 20

Case I.—*Tuberculous ulceration of the bladder. Healed.*

P. N., aged thirty-four years, a mill manager and timber cruiser, was admitted to the service of Dr. Francis J. Shepherd, October, 1906, giving the following history: (1) Swelling, pain, and tenderness in the left testicle in 1898, subsiding after four months. (2) Hemorrhages from the bladder—the first in 1904; a second in May, 1906, associated with a moderate amount of pain and frequency of micturition; a third in July, 1906; and a fourth, one week before admission. (3) With the last hemorrhage pain and frequency of micturition became much more aggravated, necessitating the evacuation of the bladder every hour or hour and a half.

The patient is a robust-looking individual, weather-beaten and ruddy. External examination is negative apart from the presence of a quiescent nodule in the left globus minor.

On November 5 a cystoscopic examination by Dr. R. P. Campbell revealed the following condition: "The mucous membrane of the base of the bladder is congested and œdematous. On the left side posterior to the trigone is a yellow adherent mass of pus 5 mm. in diameter, covering the base of an ulcer. The right ureter only was catheterized, and after washing the bladder the urine from the left kidney was simultaneously collected, as it was secreted into the bladder, by means of an ordinary catheter. The examination of the common urine taken by catheter immediately before instrumentation and the separate urines obtained in the above manner resulted as follows:

Common.	Right.	Left.
Turbid	Clear	Turbid.
1025	1027	1025.
Acid	Acid	Acid.
Albumin present	No albumin	No albumin.
Urea 2.5 per cent.	2.6 per cent.	2.3 per cent.
Pus in quantity	None	A few pus cells, possibly from bladder contamination.
Tubercle bacilli present	Ureteral epithelium, no tubercle bacilli	Tubercle bacilli present, probably from the same source as pus cells.

The first inoculation of T. R. tuberculin $\frac{1}{1000}$ milligram was given November 5. The opsonic index to the tubercle bacillus was normal.

The patient was discharged from the hospital on the understanding that he should return every ten days for treatment.

November 13. Opsonic index, 1.25. Patient reports frequency of micturition slightly less troublesome, especially during the night. The chief improvement, however, has been in the marked decrease in the pain. No further hemorrhage. Gain in weight two pounds. Tubercle bacilli in large number were again found in the urine voided. In the preparation of the slides 30 per cent. nitric acid was used as a decolorizing agent.

November 22. Opsonic index, 1.25. The patient continues to progress favorably. The bladder is generally evacuated every three hours. The time, however, is variable. The pain is distinctly less and the patient is able to walk without experiencing the discomfort from which he suffered at the time of admission. There has been a further gain in weight of two pounds. Inoculation, $\frac{1}{1000}$ milligram.

December 1. Opsonic index, 1.2. Progressing favorably.

December 11. Opsonic index, 1.18. Gain in weight since commencement of treatment eight pounds. The patient is now able to retain urine for four hours. The pain is variable, but distinctly less than at the time of last inoculation. Inoculation, $\frac{1}{800}$ milligram.

December 21. Opsonic index, 1.18. The patient continues to do well. No further bleeding; no discomfort in walking; a further gain in weight of two pounds—that is, ten pounds since the first inoculation on November 5. Inoculation, $\frac{1}{1000}$ milligram.

January 4. Opsonic index, 1.1. Total gain in weight twelve pounds. Bladder symptoms as before. Inoculation, $\frac{1}{1000}$ milligram.

January 15. Opsonic index, 0.88. Since last inoculation the patient has been confined to bed for several days with an attack of influenza. The bladder symptoms during this period were distinctly aggravated. This illness may account for the sudden decline in the index and undoubtedly is responsible for a loss of four pounds in weight. Inoculation, $\frac{1}{800}$ milligram.

January 25. Opsonic index, 1.35. The patient has noticed a marked improvement in his condition since last reporting. Net gain in weight fourteen pounds, that is, an increase of six pounds in ten days. Inoculation, $\frac{1}{800}$ milligram.

February 8. Since last reporting January 25, the patient has been perfectly well and proffers the information that the nodules in the left globus minor have diminished considerably in size, which observation is confirmed by examination. Total gain in weight eighteen pounds. Opsonic index, 1.25. Inoculation, $\frac{1}{800}$ milligram.

The bladder holds 300 c.c. of fluid without discomfort, and a second cystoscopic examination by Dr. R. P. Campbell shows the mucous membrane to be healthy throughout, with the exception of an area of œdematous swelling toward the left of the trigone and of some irregular ridges from among which the urine from the left ureter can be seen issuing. Save for this œdema and irregularity in the floor of the bladder there is nothing abnormal. No evidence is found of the former ulceration. The cystitis is confined to the œdematous area.

Comment. In the latter part of December tubercle bacilli were again found in the voided urine. The washed precipitate from a centrifugalized specimen was injected subcutaneously into a guinea-pig. Three weeks later the guinea-pig was killed, and caseous material from the glands of the groin transferred to a modification of Dorset egg medium (Duval). After five days the medium was found to be covered by a fine growth, the surface of the slants presenting the appearance of ground glass. Two tubes were opened and smears prepared, tubercle bacilli in pure culture being found. At the end of ten days transplants were made to bouillon flasks. It is our intention to prepare from the growth upon the latter a vaccine for use in the further treatment of this case.

This case has been selected from a series of three under observation. In all three improvement was marked after the first inoculation, pain being the first symptom to be relieved. In the first case of this series bilateral apical disease was associated with the infection of the urinary tract. In spite, however, of the wide distribution of the infection the patient has gained twenty pounds since the commencement of inoculations in August, 1906, with marked improvement in the urinary symptoms and pulmonary signs. The third case is of too recent date to be commented upon.

CASE II.—*Lupus vulgaris of the lip. Healed.*

W. G. G., aged twenty-nine years, medical student. About April, 1906, a tuberculous eruption appeared on the upper lip immediately below the margin of the left nostril, covering a skin area 1.5 cm. in diameter. This was treated locally with white precipitate ointment, resorcin, and other applications, all of which appeared to increase the irritation. After two months in the country some improvement was noted, but at the end of this period a large single tubercle appeared close to the original cluster. A small snipping from the original lesion was examined by Dr. Duval, who reported the condition to be tuberculous.

October 7. Opsonic index to the tubercle bacillus, 0.85. Inoculation of $\frac{1}{1500}$ milligram of T. R. tuberculin.

October 9. Opsonic index, 0.7. "Negative phase."

October 12. Opsonic index, 1.15. "Positive phase."

During October and November the eruption showed marked improvement. On February 1 the eruption upon the lip and margin of the nostril was apparently completely healed. There was a gain in weight since the commencement of the treatment of ten pounds.

Inoculations were given every ten days to two weeks, the dose since October having been $\frac{1}{800}$ milligram.

Comment. There can be no doubt as to the tuberculous nature of the eruption upon the lip, as the histological features were typical of tuberculosis and acid-fast bacilli were stained in the sections.

CASE III.—*Tuberculous arthritis and peri-arthritis of the wrist. Healed.*

M. M., aged twenty-five years. Pleurisy at eleven. Spinal caries at twelve—healed after five years' treatment. The family history is negative.

In 1901, following a slight injury, swelling of the left wrist with pain and loss of function were noted. This condition was operated upon some months later, with almost complete restoration of function. In May, 1905, the wrist again became painful and weak, loss of power in the extensors being the chief feature. The patient attended the Outdoor Department of one of the General Hospitals, where the wrist was enclosed at first in plaster-of-Paris and later in starch bandages. Subsequently she was treated by Bier's method, that is, from November, 1905, until admission to the service of Dr. Francis J. Shepherd at the Montreal General Hospital, July 18, 1906. At this time her condition was found to be unimproved.

On admission the left wrist was markedly swollen, painful, and tender, with prominence and posterior displacement of the head of the ulna and tenderness over the lower end of the radius. In front and behind the wrist-joint corresponding to the tendon areas were large pulpy swellings tender to pressure. Movements of the wrist-joint were very limited and painful. The muscles of the forearm and hand were atrophied and the fingers semiflexed at the terminal joints.

From the date of admission until August 11, Bier's bandage was applied daily without improvement. On the latter date the opsonic index to the tubercle bacillus was found to be 1.22.

The patient was given an inoculation of $\frac{1}{1500}$ milligram T. R. tuberculin. A small puncture was made with a scalpel over one of the tender points and a Klapp suction apparatus was applied for one hour, in periods of five minutes on and three off. At the same time a Bier's bandage was applied above the elbow.

This method of treatment led to almost immediate improvement and was continued until the patient was discharged December 8, 1906, four punctures in all having been made. When one puncture had so healed that the flow of serum almost or entirely ceased, a new puncture was made in another location where tenderness persisted.

The routine treatment consisted of (1) the adjustment daily of a Bier's bandage above the elbow half an hour before the application of the suction apparatus and its removal when aspiration was completed; (2) the application of the cup after swabbing the puncture area with ether and lifting off the scab with the flat end of a probe; the amount of negative pressure employed was regulated by the degree of discoloration and the sensations of the patient, the desired result being a passive hyperemia without pain; and (3) the administration of T. R. tuberculin, $\frac{1}{1000}$ milligram every ten to fourteen days.

After the removal of the suction cup a small amount of boric powder was dusted over the puncture, and dry dressings were applied with a large pad of cotton-wool to prevent injury. The patient was at all times allowed to carry out movements unassociated with pain. In this way the nutrition of the muscles was improved and every advantage secured to the joints involved against increased fixation through contraction of adhesions.

At the end of four weeks a marked diminution had taken place in the inflammatory symptoms; partial although slight movement had been restored to the wrist-joint, and the fingers no longer remained semiflexed and rigid.

January 22. Since her discharge the patient has returned at intervals of ten days for inoculation. The condition at the present time is as follows: Complete absence of pain and tenderness in the left wrist-joint. The movements of the fingers are normal and in place of their former atrophied and pointed appearance they are now well rounded. There is very slight atrophy of the muscles of the hand. The movements of the wrist-joint remain markedly restricted, confined exclusively to flexion and extension. There is distinct improvement, however, in the nutrition of the extensor and flexor groups. The patient is able to lift light articles without discomfort. The general health of the patient is better than she has ever known. Since August there has been a gain of twelve pounds in weight.

Comment. The opsonic index to the tubercle bacillus was found, shortly after admission to be about normal, 1.15. This observation substantiates the theory held by us for some months that the result obtained by Bier in the treatment of tuberculous joints by hyperemia is due to the auto-inoculation thereby secured. This theory has recently been confirmed by the observations of Patch and Wells, carried out in Wright's laboratory, upon the blood of tuberculous patients treated by this method. These observers found that during the use of the bandage the blood, if taken at frequent intervals, showed recurring fluctuations in the immunity index varying in degree with the grade and duration of the Stauung (congestion). On removal of the bandage a more marked auto-inoculation was revealed by an increased negative dip and the development of a positive phase within twenty-four hours, the index rising to 1.30, 1.40, or higher. These observers were led to the conclusion that the use of a Bier bandage should be controlled by the opsonic index, as the dose, in an auto-inoculation from different joints and at different times from the same joint in the same individual, must necessarily vary. These conclusions furnish an explanation of the frequent failures in the treatment of large joints by this method, and account for the untoward clinical phenomena following the prolonged daily use of the bandage above large joints. If such cases could be controlled by the index it would undoubtedly be found that not infrequently a "negative phase" had been engrafted upon a "negative phase" and in this way a gradual destruction of immunity units brought about. In the case reported the bandage was adjusted simply in order to secure an increased lymph tension in the diseased area, that is, an increase of lymph rich in opsonins. The use of the suction apparatus at this time not only removes stale, deopsonized serum, but promotes the inflow of more highly protective fluids into the diseased area. Further, the use of negative pressure at the time of the application of the bandage lessens the danger of superadding an auto-inoculation.

It is possible that Bier's method if controlled by the opsonic index furnishes an even more scientific method of treatment than the use of tuberculin, inasmuch as the material absorbed is the direct product of the particular strain of tubercle bacilli from which the individual is suffering, and theoretically more nearly approximates to what has been definitely established with regard to the use of vaccines prepared from staphylococci. On the other hand, the adjustment of the dose is a more difficult matter, and there remains finally the question whether the products of growth are as efficacious in arresting or retarding tuberculous disease as the actual bacterial protoplasm.

Case IV.—*Tuberculous cervical adenitis. Improved.*

Miss A., aged twenty-four years. In 1901, two glands in the right posterior triangle of the neck became enlarged, underwent softening, and were incised. The sinus persisted for over a year. In 1903 several glands in the right axilla became similarly involved, were finally opened, and continued to discharge for three years. During the period 1900 to 1903 four other glands were noticed to be enlarged, that is, the pre-auriculars and two in front of the left sternomastoid. In the spring of 1906 the submaxillary glands became infected, together with several glands in front of the right sternomastoid.

The patient was admitted to the service of Dr. J. M. Elder, at the Montreal General Hospital, September, 1906. Examination showed the presence of a complete necklet of enlarged glands, varying in size, extending from below the right ear—beneath the chin—to a corresponding point on the opposite side, with discharging sinuses behind both angles of the mandible, in the submaxillary triangles, and in front of both ears—six or seven in all. In addition, enlarged glands were palpable in both posterior triangles and in the sublingual region. One especially large gland was noted in the anterior part of the right submaxillary triangle adherent to the lower border and inner surface of the jaw. There were also palpable glands in the right axilla. The opsonic index to the tubercle bacillus was 0.82.

Owing to the extensive suppuration in the glands and tuberculous infiltration of the skin surrounding the sinuses, extirpation was considered inadvisable, and the patient was immediately placed upon inoculations of T. R. tuberculin every ten days. As Staphylococcus aureus had been secured in pure culture from the discharge the first three inoculations were combined with a vaccine of this organism containing 250,000,000 devitalized cocci to the dose. After the fourth inoculation all sinuses had completely closed and the patient showed a gain in weight of six pounds.

January 25. The patient continues to improve in general health. There has been a gain in weight of twelve pounds since commencement of treatment. All the outlying glands show a marked diminution in size. The large gland noted in the right submaxillary region is no longer tender, shows a marked decrease in size, and is quite movable.

Comment. In view of the history of prolonged suppuration associated with the earlier lesions the result obtained in this case is most striking from a clinical point of view. No doubt the prompt eradication of the associated staphylococcic infection favored an earlier inclusion of the tuberculous foci.

Case V.—*Furunculosis. Cured.*

F., aged forty years, a foreman in the washroom of the hospital laundry, has been subject to succeeding crops of furuncles on the forearms, neck, and thighs for three months, during which time active lesions have always been present. On September 5 there were three acute furuncles on the anterior surface of the right forearm, one on the left forearm in the same location, one on the back of the neck, and one on the right leg. All were severe and in the acute stage. Cultures taken from one of the furuncles on the right arm yielded in eighteen hours a luxuriant growth of Staphylococcus aureus, from which a personal vaccine was prepared.

September 6. Opsonic index to staphylococcus, 0.62. Inoculation of 500,000,000 "personal" vaccine.

September 7. Opsonic index, 0.28. "Negative phase." The local signs are more severe.

September 9. A marked improvement is noted. The furuncles are much less painful and the surrounding blush has almost entirely disappeared.

September 10. Opsonic index, 1.72. There remain only small brown crusts the size of split peas, with slight underlying induration at the site of the lesions. None of the furuncles was incised or treated locally in any way.

January 25. The patient has remained absolutely free from further infections. A second inoculation was not considered necessary.

Comment. The foregoing represents the clinical type of staphylococcic infection which yields most readily to inoculation. From the organism isolated in this case the stock vaccine of Staphylococcus aureus was prepared, which was used in Cases VII and VIII, and in the primary inoculation in Case VI.

CASE VI.—*Carbuncle. Cured.*

F. M. D., aged fifty-five years, a clergyman. A man of robust physique, but overworked for the past two years. For three months previous to inoculation he suffered from a series of carbuncles, one lesion no sooner subsiding than another developed. The patient was first seen at the request of Dr. J. C. Cameron on December 14. Some days previous swelling and tenderness appeared at the base of the right middle finger—dorsal surface. The process gradually extended with the appearance of multiple foci of suppuration and tenderness in the axillary lymph nodes.

When seen December 14 a local septic infection existed at the base of the right middle finger with signs as above described. Owing to the patient's serious condition a 500,000,000 dose of staphylococcic vaccine was administered. A specimen taken from the finger for the determination of the opsonic index revealed a very pale, fluid condition of the blood; opsonic index to staphylococcus 0.58.

December 15. Agar slants from the carbuncle yielded pure cultures of Staphylococcus aureus.

When the patient was seen twenty-four hours after inoculation his general condition was found to be much improved. The axillary glands were free from tenderness. No further complaints of pain were made and the damaged finger could be handled without discomfort.

On December 17 the finger was quite normal apart from a moderate amount of induration and discoloration at the site of the lesion. Movements at the metacarpophalangeal and first interphalangeal joints were completely restored. Opsonic index, 1.28. The patient constantly reiterated his astonishment at the rapidity of his recovery.

On December 27 there were no signs of further suppuration. Opsonic index, 2. The patient, however, was given a second inoculation of 500,000,000 of "personal" vaccine as a precautionary measure.

February 1. The patient remains free from further infections.

CASE VII.—*Carbuncle. Cured.*

D. L., an Italian, aged fifty years, was first seen in the Outdoor Surgery December 7. Examination showed the presence of an area 10 x 10 cm. over the right scapula intensely inflamed and presenting numerous foci of suppuration. This area was also traversed by a crucial incision made two days previously. The discharge was profuse. In the interscapular area were numerous furuncles in the acute stage. There was in addition an area of induration 10 cm. in diameter on the right side of the posterior surface of the neck embracing three distinct foci of suppuration, from one of which Staphylococcus aureus was isolated in pure culture. In order to save time, however, the patient was at once given an injection of 500,000,000 of "stock" vaccine.

The patient was not seen again for seven days, at the end of which time all signs of inflammation had disappeared from the lesions above described. There remained only a healthy granulating ulcer occupying the site of the crucial incision. Opsonic index, 1.22. Further inoculation was not deemed necessary.

Case VIII.—*Suppurative periostitis. Cured.*

J. S., aged forty-five years, was seen in the Outdoor Department September, 1906. Examination showed a large fusiform swelling of the left middle finger with a sinus over the palmar surface of the middle phalanx. Denuded bone could be felt with a probe. The finger was extremely painful and tender. Gram positive cocci were found in smears. Opsonic index to staphylococcus, 0.75. An inoculation of 500,000,000 of Staphylococcus aureus vaccine was given.

Three days later the patient was again examined and the following condition noted: Marked diminution in the size of the finger. Complete absence of pain and tenderness. Complete absence of discharge. At the bottom of the sinus denuded bone could be seen. Although enjoined to report daily the patient failed to return for subsequent observation.

Later this patient was seen by Dr. Hillman, resident in charge of the Outdoor Surgery, who examined the finger and found it to be normal in appearance and function. The patient stated that he had not returned, as the sinus had healed rapidly without suppuration or separation of bone.

Case IX.—*Multiple abscesses of the axilla. Cured.*

L. C., aged eighteen years, was admitted to the Montreal General Hospital, service of Dr. J. Alex. Hutchison, September 12, 1906. The patient was in good health prior to September 4. The first symptoms were stiffness and pain in the region of the right shoulder associated with chilly sensations. It was noticed that the shoulder was swollen and that a hard lump about the size of an egg could be felt in front of the joint.

On admission the patient was a well-nourished, anemic adult. A fluctuating inflammatory mass about the size of an egg was felt beneath the margin of the pectoralis major muscle. Cellulitis of the skin and subcutaneous tissue extended over the front and outer aspect of the shoulder and down the arm to the level of the insertion of the coracobrachialis muscle.

September 19. Incision of abscess with multiple incisions of the skin.

October 1. Wounds practically healed. About this time a number of pustules appeared in the vicinity of the cicatrix and also on the inner side of the arm. These were incised. One week later a hard mass appeared in the axilla about the size of a walnut. This was incised and pus evacuated.

During the following three weeks other abscesses continued to form in rapid succession in the superficial tissues of the axilla and were opened as they appeared, sometimes as many as two or there in a week. Owing to the serious impairment of the patient's general health and the progressive loss of function in the arm the patient was placed upon inoculations.

Agar slants from discharge yielded pure cultures of Staphylococcus aureus, from which a vaccine was prepared. First inoculation, November 22, of 500,000,000. Opsonic index to staphylococcus, 1.1.

October 26. Opsonic index, 1.5. A marked improvement is noted. The area of induration is very much lessened and all sinuses have healed.

October 29. Dressings discontinued as all signs of inflammation and suppuration have disappeared. There is complete restoration of the movements of the arm.

December 1. Opsonic index, 1.4. The patient still remains free from further suppuration.

January 8. The patient returned today complaining of a recurrence of tenderness in the axilla. Examination revealed the presence of a hard indurated mass the size of a filbert in the axilla near the posterior fold. Inoculation, 500,000,000. Arm bandaged without incision. Opsonic index, 0.70.

January 22. The patient has been seen several times since the last note was made and local examinations have always been negative.

February 1. The general health is greatly improved and the patient states that he has gained in weight twelve pounds.

Comment. This patient received in all five inoculations, although all symptoms disappeared after the first injection, with the exception of the slight disturbance observed on January 8, which it is interesting to note was coincident with a marked decline in the index.

CASE X.—*Acne indurata. Cured.*

S., aged twenty-three years, a medical student, has had acne indurata of the face for the past four years. He was seen first on October 10, 1906, at which time there were numerous active pustules present upon both cheeks, with extensive scarring. His general health was good. Various methods of treatment had been tried, but without even temporary improvement.

October 12. The patient was inoculated with 500,000,000 staphylococcus albus "personal." Opsonic index to staphylococcus normal.

October 22. A marked improvement is noted. No new pustules. Opsonic index, 1.7. A second inoculation of 500,000,000 was given.

Following this the patient had a severe negative phase with an increase in the local inflammatory signs which persisted for six or eight days and were followed by a second period of improvement.

December 10. No further pustules have developed. The skin of the cheeks has become much paler than at any time previously. Further inoculation was not considered necessary.

December 18. The patient still remained absolutely free from pustules and expressed great delight at the result achieved. A further inoculation of 250,000,000 was given as a precautionary measure.

February 1. There has been no further pustulation and the skin of the cheeks has become soft and pliable. The purplish discoloration in the scars has almost entirely disappeared except when exposed to cold.

Comment. Several cases of this affection have been under observation and the results obtained have varied directly with the duration of the disease. While long-standing cases (six to eight years) show marked improvement after the first inoculation it is necessary to continue the treatment over a much longer period of time; apparently the presence of the scar tissue with the attendant defects in the circulation plays an important role in delaying a cure. We have found a valuable adjunct in the management of such cases in the active hyperemia induced by the application of hot-water stupes for a period of twenty minutes daily. By this means the defects in the circulation are overcome. Further, it has been noted that after inoculation in old-standing cases the local signs are more prone to exacerbations, necessitating not infrequently a reduction in the dosage.

As the treatment of acne indurata is always more or less prolonged we have been able on several occasions to make observations upon the effect of inoculation on the viability of organisms in unopened pustules, and have repeatedly obtained during the positive phase pus from apparently active lesions which yielded negative results upon ordinary media.

Our experience with the use of stock vaccines in the treatment of such cases has been unsatisfactory, confirming in this respect the writings of Wright, Weinstein, and others upon the importance of preparing in each case a personal vaccine.

Without exception the organism isolated in these cases has been Staphylococcus epidermidis albus (Welch), yielding upon plain agar within twenty-four hours medium-sized china-white colonies of Gram-positive cocci.

CASE XI.—*Gonorrhœal polyarthritis. Cured.*

W. M., aged twenty-one years. Admitted to the Montreal General Hospital, service of Dr. Finley, December 10, 1906, with a history of urethral discharge dating from December 1, and complaining of severe pain and swelling in the right shoulder, left wrist, and metacarpophalangeal joint of the left thumb. The joint symptoms manifested themselves simultaneously on December 6, in the three joints described; the pain and swelling reached their present degree of intensity within forty-eight hours.

Examination revealed the presence of: (1) A profuse urethral discharge, which on examination was found to contain numerous intracellular diplococci decolorized by Gram's stain. (2) An acute arthritis of the right shoulder, left wrist, and metacarpophalangeal joint of the left thumb.

From the date of admission until December 24, the joint lesions were treated locally with fomentations of lead and opium, without improvement. During this period the temperature ranged daily between 99.3° and 101.3° F.

December 25. The patient was given an inoculation of stock gonococcus vaccine—10,000,000. Opsonic index to gonococcus, 0.85

December 26. The patient states that he feels somewhat better. No improvement is noted in the joint condition.

December 27. Opsonic index, 3. A most striking change has taken place in the arthritic lesions; the patient freely moves the left hand and abducts the right arm to a right angle with the body with rapidity and ease. Temperature range, 98.1° to 99°. There is a very marked decrease in the urethral discharge.

From this date the joint symptoms rapidly abated, and when the patient left the hospital January 2 he was able to carry out the fullest movements in the shoulder and wrist-joints without pain.

On January 7 the patient resumed his occupation as typewriter.

A second inoculation of 10,000,000 was administered on January 4 and a third on January 14. On the latter date the urethral discharge had entirely ceased.

February 8. The patient has remained absolutely free both from articular and urethral symptoms.

Comment. Our attention was first drawn to this infection by the striking evidence of auto-inoculation uniformly manifested during the acute stage of gonorrhœal epididymo-orchitis in the sudden cessation of the urethral discharge. With the backward extension of the disease and the attendant inflammation there takes place an absorption of gonococcus toxins—in some cases of the organisms themselves—leading as we have found to a marked rise in the immunity index, which latter feature is responsible in our opinion for the marked decrease or complete disappearance of the urethral discharge; an attenuated discharge reappearing in the majority of cases with the decline of the complicating inflammation and the concurrent ebb of the immunity. The index seldom, however, falls below normal.

Prompted then by the results of auto-inoculation interpreted along the above lines, three patients with acute gonorrhœa, complicated by polyarticular inflammation, have been subjected to inoculations of gonococcic vaccine. In all three the effect upon the urethral discharge was identical with the phenomena to be observed in epididymal involvement: the cessation of the discharge following the development of a strong positive phase. The following indices were recorded: Case I, forty-eight hours after inoculation, opsonic index, 1.6. Case II, forty-eight hours after inoculation, opsonic index, 2.8. Case III, twenty-four hours after inoculation, opsonic index, 3. The last is the case reported. Case I received a second inoculation and was discharged well. Case II was admitted from the Out-patient Department owing to extensive effusion into the knee-joints and remained under observation for four months owing to the persistence of the effusion in the right knee, leading eventually to partial fibrous ankylosis. Fluid aspirated from the right knee on two occasions was sterile and no organisms were found in smears.

CASE XII.—*Epidemic cerebrospinal meningitis. Recovery.*

L. B., aged twenty-eight years. Admitted to the Montreal General Hospital December 21, service of Dr. F. G. Finley, complaining of severe pain in the back of the head, supra-orbital regions, and the small of the back, and also of chilly sensations alternating with profuse sweats.

The patient was in good health until December 17, when he contracted what he describes as a severe cold, and felt "all choked up in the head." From December 18 to the time of admission pain in the head had been constant. There were sharp pains in the occipital region radiating forward to the supra-orbital regions. Additional symptoms were chilly sensations, frequent sweats, and occasional vomiting.

On admission he was well nourished; the skin was hot and dry; mucous membranes were of good color. The patient felt chilly and suffered from severe paroxysms of pain in the back of the head which caused him to cry out and groan. The tongue was coated. The bowels were constipated. The abdomen was normal. The urine was clear, acid, 1041, no albumin, 1.54 per cent. sugar. Microscopically, a few pus cells and mucous threads.

The mind was clear. There was no retraction of the neck, but the patient was unable to approximate the chin to the sternum and any attempt to pull the head forward caused great pain. Kernig's sign was present on both sides, and there were slight fibrillary twitchings in the left calf muscles. The plantar reflexes and the knee-jerks were increased. Achilles, cremasteric, abdominal, epigastric and supra-orbital reflexes were marked.

December 22. Temperature rose from 99° at noon, to 103.6° at 4 P.M., with general symptoms as before noted. At 12 M. lumbar puncture was performed and 1.5 c.c. of turbid fluid withdrawn, containing numerous polynuclear neutrophilic cells. A few intracellular diplococci decolorizing by Gram were found. At 6 P.M. the patient was given hypodermically an injection of meningococcic vaccine containing 15,000,000 devitalized organisms. The patient's opsonic index to the meningococcus taken at the same time was found to be approximately normal.

December 23. Temperature range 101° to 102°. The headache has been less intense. There is profuse sweating at irregular intervals. Cultures from the spinal fluid upon blood agar show at the end of twenty-four hours numerous colonies of meningococci.

December 24. Opsonic index, 1.5. Temeprature range, 100° to 102°. The headache continues, but is less severe. There is constant ache with pain on pressure in the muscles of the lumbar region. Kernig's sign is still present, but the angle of extension is greater, 135° on both sides. Sugar in the urine, 0.48 per cent.

Examination of the eyes by Dr. George M. Mathewson shows "optic neuritis in both eyes—distinct but of moderate degree. No hemorrhages or white masses in the retinæ."

December 27. The patient complains only of a slight pain in the back. The right pupil is dilated, the left slightly contracted; both react to light. Reflexes normal. The patient is able to bring the chin close to the sternum without much pain. Kernig's sign is still present. Temperature range 98° at 8 A.M. to 100° at 4 P.M. General condition much improved. Sugar—a trace only.

January 1. Kernig's sign is absent; all reflexes are normal. The right pupil is still a little larger than the left. No pain in the back or head. The patient is feeling much better in every way. No sugar.

January 3. Opsonic index, 5.9. The patient feels and looks well. Temperature range for the last twenty-four hours, 97.4° to 99.4°. No headache or pain in the back. Very little rigidity of the muscles of the neck. Reflexes normal, pupils equal and active. Further inoculation not considered necessary.

Comment. Attention is directed to the following points in connection with this case: (1) It is scarcely probable that the small amount of fluid removed (1.5 c.c.) could have influenced the course of the disease. Temporary improvement is undoubtedly seen after spinal puncture, generally when larger quantities than the above are removed. In the present instance further puncture, though under consideration, was postponed from day to day owing to the patient's steady progress toward recovery. (2) Glycosuria was found as a rare concomitant and it is interesting to note that with the rise in the index the glycosuria rapidly disappeared. (3) Exacerbations so frequently observed in the cases of moderate intensity were not noted in the present instance. (4) An extremely high immunity index was noted on January 3. In view of the results obtained by Flexner in his experimental work upon the protective powers of the serum of immunized monkeys this was not unexpected, and in our opinion was due directly to the immunity induced by inoculation, as observations made in June, 1906, upon the serum of a patient convalescent from subacute epidemic cerebrospinal meningitis yielded indices below the normal.

Case XIII.—*Epidemic cerebrospinal meningitis. Recovery.*

A. M., aged seven months. Admitted to the service of Dr. F. G. Finley, at the Montreal General Hospital January 28, 1907. The present illness began January 15, with fever, pain referred to the neck, and retraction of the head. The temperature rose rapidly to 105° and subsequently ranged between 100° and 104° until admission. Neither vomiting nor convulsions occurred. Bowels constipated. On admission the patient was a well-nourished Hebrew child. He lies upon the side with legs and thighs flexed. The head is retracted and rotated to the right and attempts at manipulation cause great distress. Temperature at 2 p.m., 104.2°.

Shortly after admission lumbar puncture was performed and 45 c.c. of turbid fluid withdrawn. Cover-slip preparations showed numerous meningococci, both free and intracellular. Cultures were positive at the end of twenty-four hours. The opsonic index to meningococcus was 0.68. Inoculation of meningococcic vaccine, 7,500,000.

January 31. Opsonic index, 1.8. Temperature range, 99.2° to 103°. The child is steadily improving.

February 5. Opsonic index, 1.58. Temperature range, 100° to 102°. Inoculation, 7,500,000. The child continues to progress satisfactorily.

February 6. Temperature range, 100° to 100.6°. The child is much better in every way.

February 8. Opsonic index, 1.2. From this date until February 11 the temperature ranged between 98.2° and 99.1°. On February 11 the temperature rose from 99° at 8 p.m. to 102° on the morning of February 12, with return of cardinal symptoms. Inoculation, 7,500,000.

February 13. Temperature range, 97.4° to 98.1°. The child is apparently convalescent.

February 15. Opsonic index, 2.8. A final inoculation of 15,000,000 was given. From this date convalescence was uninterrupted.

Comment. In this case the notes have been drawn to accentuate the effect of inoculation upon the temperature range. The second lumbar puncture was performed on January 31, as a result of the index finding, in the hope that the removal of deopsonized fluid would favor the inflow of a lymph approximating in opsonic power the individual's blood serum. When large quantities of fluid are removed by lumbar puncture the part played by the replacing lymph must come to be considered in the light of an auto-inoculation. It is our opinion that in the management of these cases lumbar puncture should be practised at frequent intervals in order to rid the spinal lymph space of fluid incapable either of destroying or inhibiting the growth of the infective agent. That the cerebrospinal fluid withdrawn in such cases has been reduced in opsonic power is shown by the fact that incubation increases very considerably the number of diplococci present. Upon this important point we are at present at work and hope shortly to publish our results in full.

CASE XIV.—*Epidemic cerebrospinal meningitis. Death. Autopsy.*

H. G., aged forty years, a jeweller. Admitted to the service of Dr. Molson, at the Montreal General Hospital, February 28, 1907.

The present illness began February 13, with severe chill, intense headache, and stiffness of the neck developing within twenty-four hours. Vomited once.

February 16. The patient no longer recognized his family, remaining in this semiconscious state until admission.

On admission the patient responds to repeated requests to turn in bed or protrude the tongue, but is unable to speak. The face is expressionless. Movements of the head cause intense pain. Kernig's sign is marked. The temperature is subnormal.

March 1. Lumbar puncture was performed and 15 c.c. of turbid fluid was withdrawn, containing numerous meningococci, both free and intracellular. (Twenty-four-hour cultures positive.) Inoculation of meningococcus vaccine, 15,000,000.

March 3. Condition unchanged. Muttering delirium is noted at times, with frequent picking at the bed-clothes. Temperature at 12 M., 102°.

March 6. The patient is quite unconscious and cannot be aroused. Temperature, 98.2°; pulse, 60. Inoculation, 30,000,000.

March 7. Condition unchanged. Temperature, 97.4°.

March 8. Inoculation, 30,000,000. Lumbar puncture performed and 10 c.c. of fluid withdrawn, containing meningococci as before. (Twenty-four-hour cultures positive.)

March 9. The patient's mental state today shows a marked improvement. He answers questions rationally, looks bright, and is able to take feeder from attendant. Rigidity of the neck and extremities is still marked. Temperature, 100°.

March 10. The patient's mental condition continues to improve.

March 11. It is difficult to arouse the patient this morning; nourishment is not so well taken and the expression is again lethargic. At 12 M. the patient became cyanosed, and died at 3 P.M.

Extract from autopsy protocol. Thirty-six hours after death (Dr. Duval). The longitudinal sinus contains fluid blood. On stripping back the dura the brain substance is quite dry and the convolutions markedly flattened. Pial vessels are deeply injected. At the base of the brain in the region of the pons there is considerable opacity of the meninges, while in places there is a dry, plastic exudate. Brain weight, 1200 grams. The lateral ventricles are greatly dilated and filled with slightly turbid fluid in which there is some fibrin. The Sylvian canal is plugged with this fibrinous material. The fourth ventricle appears normal. The pons, medulla, midbrain and basal ganglia show nothing remarkable. The hemispheres, the optic thalami, and the striate bodies are more or less œdematous. In the cover-slip preparations from the exudate in the pontine region and from the ventricular fluid no diplococci were found. Cultures negative.

Comment. We would draw attention to the fact that while viable diplococci were present in the fluid withdrawn from the spinal canal three days before death the autopsy findings showed a complete arrest of the meningitis. Unfortunately the lapsed time between death and the postmortem examination detracts greatly from the value of the bacteriological observations.

While we do not presume to draw conclusions from the treatment of so few cases we certainly look forward to the acquisition of additional evidence upon the value of bacterial inoculation in this affection, both in the field of protection and of cure.

GENERAL CONCLUSIONS. Our observations confirm in general the dictum of Wright that an individual suffering from a localized infection has a low opsonic index to that particular infection. We have found low indices to be the rule in afebrile tuberculous lesions, in furunculosis, and in acne during the earlier stages. Long-standing cases of acne have been found in our experience to yield indices between 0.90 and 1.1. In this connection the uniformly low indices noted by Weinstein are remarkable.

When the lesions are exposed to view, as in lupus, furunculosis, and acne, the first effect to be noted is a rapid subsidence of the inflammatory signs, which explains the early diminution in the subjective symptom of pain in such deep-seated lesions as tuberculous ulceration of the bladder, tuberculous arthritis, and meningitis. This relative anemia is invariably associated with the development of a positive phase just as increased congestion is, in the majority of cases, the concomitant of a negative phase.

While the opsonic index is a most valuable guide in regulating the amount and time of dosage, we are of the opinion that exellent results may be obtained, especially in the treatment of localized tuberculous lesions, without the use of the "Immunity Thermometer;" success in the vast majority of cases attending the employment of minute doses at long intervals, the dose beginning preferably with $\frac{1}{1500}$ milligram to be gradually increased to $\frac{1}{800}$ milligram, providing always that increases are unattended by constitutional symptoms. Generally speaking the feelings of the patient are a safe guide. Trudeau persisted in the use of tuberculin throughout the period when tuberculin was least in favor, and with most encouraging results, the guiding principles in his use of it being minimal dosage and wide spacing.

In our experience "an accumulation in the direction of a positive phase" is not to be looked for in the treatment of tuberculous cases.

The same remark applies to staphylococcic infections. Generally speaking the most that can be hoped for in these infections is a maximum in the direction of a positive phase and a minimum in the direction of a negative phase. On the contrary, in such infections as gonorrhœa and epidemic cerebrospinal meningitis high indices may be produced by inoculation, and from the striking effects of inoculation observed in gonorrhœa and the analogy which exists between the two infections, we are emboldened to hope that in bacterial inoculation may be found the form of therapeutics destined to combat effectively meningococcal infections.

TECHNIQUE. No pains have been spared to make the technique in connection with the determination of the indices as perfect as possible. Sera are collected at the same hour each day and the controls taken at the same time. Washed corpuscles are always prepared immediately before use, as we have found that in those which have stood for some time many of the polynuclear neutrophiles fail to functionate. In counting we determine the content of one hundred cells, and in the case of tubercle prepare slides from two control sera, the average being taken as the normal standard. In this determination the counts have on some occasions been exactly similar—the variation as a rule not exceeding the average per cell.

We prepare our tubercle suspension daily by rubbing up in an agate mortar a small portion of an emulsion of tubercle bacilli with 1.5 per cent. NaCl solution, centrifugating, and drawing off and thoroughly mixing the supernatant fluid. The strength of the suspension thus obtained is determined by putting through a "trial trip" with control serum and subsequently diluting the suspension so that the cell average may not exceed 4 to 6. Suspensions thus prepared and standardized rapidly deteriorate through clumping, and for this reason we are forced to the laborious undertaking of preparing daily a fresh suspension.

In preparing suspensions of other organisms 0.85 per cent. NaCl solution is employed, young cultures are used, and the density is determined by the naked-eye appearance.

In conclusion we wish to express our gratitude to Dr. Duval, Director of the Laboratory of the Montreal General Hospital, for his very special assistance and advice in connection with the bacteriological side of our work. Our thanks are also due to the members of the attending staff who have placed material at our disposal, and to Sir Almroth E. Wright for the opportunities enjoyed in his laboratory at St. Mary's Hospital, and for his encouragement of our efforts during his recent visit to Montreal.

THE OPSONIC INDEX IN DIABETES MELLITUS.[1]

A PRELIMINARY RECORD OF THE FINDINGS IN 22 CASES OF GLYCOSURIA, WITH REMARKS ON THE TECHNIQUE OF THE OPSONIN TEST AND ON ITS CLINICAL UTILITY.

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The objects of this paper are twofold: (1) To present a preliminary record of the behavior of the opsonic index in diabetes mellitus; and (2) to draw attention to certain errors and technical difficulties in opsonin testing—drawbacks which may keenly discourage the beginner, and even in the experienced worker at times arouse skepticism about the real value of the test. The experiments herewith reported were made, with the help of my co-worker, Dr. E. J. G. Beardsley, in the clinical laboratory of the Jefferson Medical College Hospital.

In selecting diabetes mellitus as a disease not unlikely to show deviations from the normal opsonic values, we were influenced primarily by the undue susceptibility of many diabetics toward bacterial infections, notably of the cutaneous and pulmonary systems —a tendency perhaps to be explained by a low opsonic content in the blood plasma of the person thus affected. Furthermore, the grave metabolic disturbances of diabetes, leading, for example, to hyperglycemia, to lipemia, and to the accumulation of β-oxybutyric acid in the blood and other tissues, prompted, on *a priori* grounds, the query, "How much, if at all, do these changes influence the blood opsonins?"

THE OPSONIC INDEX IN GLYCOSURIA.

Of the 22 patients studied, 16 were examples of diabetes mellitus, 4 of diabetes insipidus, and 2 of transient, "accidental" glycosuria. Staphylococcus pyogenes aureus was chosen as the bacterium with which to determine opsonin values in all the tests, since it is to infections from this source that the diabetic appears to be highly vulnerable. The following data, therefore, relate to the opsonic indices toward the staphylococcus alone.

Of the cases of true diabetes mellitus, all had subnormal opsonin indices, ranging between 0.34 and 0.72, and averaging 0.62 for the series of 16 patients. The individual figures for these cases were, consecutively, as follows: 0.66, 0.44, 0.68, 0.49, 0.52, 0.67, 0.59, 0.59, 0.60, 0.34, 0.72, 0.46, 0.67, 0.62, 0.46, and 0.49. On analysis of the histories of the foregoing cases, it was found that 3 in which the urine gave positive tests for acetone and diacetic acid showed especially low opsonin values: 0.49, 0.52, and 0.34 in Cases IV, V, and X, respectively. These findings, though suggestive, should not, on such meagre data, be interpreted as indicating that acidosis especially lowers the opsonic index; on the contrary, extremely subnormal values may occur in the absence of these urinary changes, as in Cases II (index, 0.44), XII (index, 0.46), XV (index, 0.46), and XVI (index, 0.49), in none of which was acetone or diacetic acid detected. In the cases thus far studied no relationship between the sugar content of the urine and the opsonic value of the blood could be determined in the individual instance, low opsonic figures having been met with in patients with moderate glycosuria, and *vice versa.* Albuminuria seemed also to have no effect upon the subject's opsonicity. No relationship could be established between the leukocyte count and the opsonic content of the serum, and the same was true of the hemoglobin and the erythrocyte figures. This bare statement will be substantiated when sufficient data have been collected to justify a more detailed discussion of the subject. Here may be noted, however, Bulloch and Ledingham's experiments[2] which showed that the injection of tallianin, though it promptly excites leukocyte increase, fails to raise the opsonic index; while in many of the patients to whom Huggard and Morland[3] administered yeast, in their studies of its effect in tuberculosis, subnormal leukocyte counts were found to be compatible with high opsonin figures and the reverse of this was likewise true.

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[1] Read before the Section on General Medicine of the College of Physicians of Philadelphia, March 11, 1907.

For the 4 cases of diabetes insipidus examined, indices of 0.82, 0.82, 0.87, and 0.89 were calculated, these figures being within the normal range, which varies between 0.8 and 1.2, according to Bulloch's studies of 84 healthy subjects.[4] The average index of these cases (0.85) is a decided contrast to that of diabetes mellitus (0.62), though it is of course obvious that the former is based upon too small a number of tests to be comparable with the latter. It may also be noted that the indices found in diabetes insipidus, though individually within the range of health, collectively average some ten points below the average normal figure, which is generally conceded to be 0.95.

Of the 2 patients with transient glycosuria, 1 showed an index of 0.81 and the other an index of 0.91; the former was an example of postoperative glycosuria, while in the latter the patient's temporary glycosuria was traced to overindulgence in malt liquors.

In view of the numerous and convincing studies of opsonins made during the last year or two, it need hardly be explained that the presence of these substances in the blood serum is essential to the display of phagocytosis by the cells concerned in this defensive process against disease due to bacterial invasion of the human organism. This impotence on the part of the leukocytes to engulf microörganisms, unaided by the opsonic substances of the blood, has been decisively proved by Wright,[5] Douglas, Bulloch,[6] and others, who have shown that after the incubation of equal units of a bacterial emulsion, washed leukocytes, and blood serum which has been heated to 60° C., the leukocytes of the mixture contain no bacteria whatever; on the other hand, if unheated serum be employed in a precisely similar test, the leukocytes are found to be crowded with bacteria which these cells have engulfed and are digesting. The inference from such an experiment is that the failure of the cells to act as phagocytes in the first instance and their ability to do so in the second, is referable to the influence of opsonic bodies, which are thermolabile, and to all intents and purposes disappear from the blood serum after it has been heated for a short time at a temperature of 60° C. It is believed that the opsonins combine with the invading bacteria and in some manner cripple them so as to favor their ingestion by the phagocytic leukocytes, which, without this preparatory attack by the opsonins, are incapable of becoming bacterial destroyers. Thus, while opsonins do not directly stimulate the leukocytes to exert bactericidal properties, they bring about such an action by their effect upon the offending microörganisms. The opsonic index of a given blood may be regarded as the capacity of its serum to stimulate phagocytosis, for the higher the opsonic content of the serum the more active the power of its leukocytes to engulf bacteria. In passing, it is of interest to mention the prevalent belief, that the blood serum contains different opsonic substances, each of which has selective combining properties with certain bacteria—an adequate opsonic index toward the staphylococcus does not mean an equal power of repelling invasion by the streptococcus, the pneumococcus, or the gonococcus. That the specificity of opsonins is more than a convenient hypothesis seems true, from the behavior of these substances in an advanced case of pulmonary tuberculosis, in which, despite the grave inroads of the infection, the patient's blood may show a high index toward the tubercle bacillus and a low index toward the streptococcus. This suggests not only a multiplicity of opsonins, but also indicates an adequate acquired resistance toward Koch's microörganism linked with a feeble opposition toward the streptococcus, to the secondary invasion of which most of the late severe symptoms of phthisis are referable.

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[2] Lancet, 1905, ii, 1603.

[3] Lancet, 1905, i, 1493.

[4] Trans. Path. Soc. Lond., 1905, lvi, 334.

This inquiry establishes merely the fact that diabetics show feeble resistance toward staphylococcic infections, if one may gauge this property by the behavior of the opsonic index toward the specific bacterium of such processes. If this be true, it may explain the prevalence of boils, carbuncles, abscesses and similar lesions in this disease, but it is premature to assert that all subjects thus affected show especially wide deviations from the normal standard, despite the apparent obviousness of this relationship in an occasional instance. The existence of a high opsonic figure in a subject of furunculosis suggests that the individual's resisting powers are being vigorously exerted. The opsonic activity of diabetic blood toward the streptococcus, the tubercle bacillus, and other microörganisms remains to be investigated, and these studies will be the subject matter of a subsequent report.

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[5] Proc. Roy. Soc., vol. lxxii, p. 357.

[6] Loc. cit.

Theoretically, it is convenient to assume that the subnormal opsonic values in diabetes are due to an inherent deficiency of opsonins, or to the antagonistic action of anti-opsonins. The latter substances neutralize the activity of opsonins without injuring the leukocytes, as shown by the experiments of Hektoen and Reudiger[7] on the anti-opsonic action of calcium chloride, barium chloride, lactic acid, and other substances. May not antiphagocytic bodies of this sort arise in consequence of the metabolic disturbances so intimately a part of the diabetic state? It may be that further investigation will establish some relationship between the opsonic content of the blood plasma and the hormon bodies, of which the substance "secretin" apparently is a real factor of certain forms of diabetes. If the admirable studies of Bayliss and Starling[8] be accepted, secretion is produced by the action of dilute acids (*i. e.*, acid chyme) on the duodenal mucosa, whence it is carried by the vessels to the pancreas upon which it acts as a stimulant to the secretion of pancreatic juice. It has also been suggested that secretin has the same effect on the internal secretion of the pancreas, and if this be true, its influence on glycosuria is obvious, granted, of course, that the pancreas is not too extensively diseased to respond to stimulation. In this connection may be noted Bainbridge and Beddard's report[9] on the finding, in 5 of 6 cases of diabetes without pancreatic lesions, minimal quantities of secretin in comparison with the amount of this material discovered in subjects dead of non-diabetic diseases.

OPSONIN TECHNIQUE.

It is obvious to anyone who has worked with the opsonin test that satisfactory results demand considerable patience, a certain manual dexterity, and an absolutely faultless technique on the part of the examiner, for the lack of which one's theoretical conception of the subject, however thorough, and the laboratory equipment at one's command, however elaborate, can never compensate. On paper, the test is a simple procedure; but in the laboratory it necessitates most rigid adherence to each technical detail, from the collection of the blood sample until the stained specimens are placed on the stage of the microscope. One false step may be sufficient to negative the result of an hour or two of continuous work. Briefly, the opsonic power of any given blood is ascertained by comparing the phagocytic activity of its serum with that of a normal serum used as a control, the resulting figure being known as the "opsonic index" of the blood in question. This is accomplished by mixing in a pipette equal parts of a bacterial emulsion, washed leukocytes, and the serum to be tested; incubating this mixture for a definite period at body temperature; and, finally, counting the bacteria ingested by a certain number of the leukocytes. Two tests are made in this manner: one with the patient's serum and the other with the serum of a healthy person, to serve as a control, the same bacterial emulsion and washed leukocytes being, of course, employed for both.

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[7] Jour. Infect. Dis., 1905, ii, 129 and 141.

[8] Jour. Amer. Med. Assoc., 1907, xlviii, 524.

[9] Biochem. Jour., 1906, i, 429.

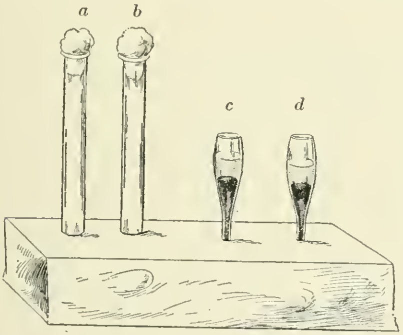

Fig. 1.—Test tube rack: *a*, bacterial emulsion; *b*, washed leukocytes; *c*, serum to be tested; *d*, control serum.

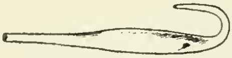

Fig. 2.—Wright's tube.

Fig. 3.—Test tube.

The details of this process, with its common sources of error, may be conveniently described under these headings:

I. Collecting the blood and separating the serum.
II. Preparing the bacterial emulsion.
III. Obtaining the washed leukocytes.
IV. Measuring, mixing, and incubating the units.
V. Preparing and examining the stained films.

Special Apparatus The special apparatus required for opsonin work comprises collecting tubes, test tubes for the washed leukocytes and for the bacterial emulsion, capillary mixing pipettes, and ordinary suction pipettes. A wooden rack (Fig. 1) for holding the several liquids will prove most advantageous in the procedure of measuring the units. Wright's tubes (Fig. 2) are by far the most satisfactory device for collecting the patient's and the control bloods. They are fashioned of glass tubing, which is drawn out at one end into a straight point not more than 1 mm. in diameter, while the other extremity is bent in the form of a *J*, the caliber of which should be of capillary fineness. The length of such a tube, from bend to straight end, is 6 cm., its greatest diameter is 5 mm., and its capacity is 1 c.c. Test tubes (Fig. 3), used in obtaining the washed leukocytes, hold 4 c.c., have a length of 7 cm., and a diameter of 9 mm.; the latter measurement is important, for it is difficult to fill a smaller-sized tube, and with a larger caliber the zone of leukocytes thrown down by centrifugalization is so shallow that it can be pipetted off only with great difficulty and much uncertainty. Tubes of the same dimensions are of a convenient size for containing the bacterial emulsion. The mixing pipettes (Fig. 4) are made of 5 mm. tubing, preferable with a relatively thick (0.5 mm.) wall, in order to avoid undue breakage. One end is drawn out into a very fine bore 15 cm. in length, and to the other end is fitted a soft rubber nipple, which may be attached directly to the larger end of the pipette, or coupled to it by a throttle and two short lengths of rubber tubing, if it be considered advisable to ensure better control of the columns of liquid while measuring the units of bacteria, leukocytes, and serum. A throttle suggested by Wright (Fig. 5) consists of an outer glass shell 5 mm. in diameter at each end, and constricted in the middle where it encloses a fine needle-like capillary tube, held in place by sealing wax, with the pointed end toward the nipple. A device of this sort has been applied by Boggs[10] to the hemocytometer, and its use in opsonin estimations is of distinct service, should one have difficulty in measuring accurately the units with the aid of a plain nipple. In lieu of such a device, a plug of cotton may be thrust into the pipette between the expansion and the "nipple end," to control the suction force. A handy form of suction pipette (Fig. 6) for removing the liquid from the washed leukocytes may be of a bit of 5 mm. tubing, about 15 cm. long and provided with a central extension 10 or 12 mm. in diameter; a similar tube is also helpful in removing the bacterial emulsion after centrifugalization. These pipettes, it may be added, are not absolutely essential, for ordinary large medicine droppers may be used instead. A few glass watch crystals, covers, and slides comprise the other glassware used in the tests. Experience has taught that for rapid and accurate work a high-speed electric centrifuge is indispensable for obtaining the washed leukocytes and for separating the serum from the whole blood without delay. In the Jefferson Clinical Laboratory, we use a centrifuge revolving 8000 times a minute, driven by a $\frac{1}{12}$ horse-power motor supplied by a 110-volt direct street current. Water-power centrifuges, which give at the most but 1000 or 1200 revolutions a minute, have not proved satisfactory in our hands.

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[10] Jour. Amer. Med. Assoc., 1907, vol. xlviii, p. 49

I. *Collecting the Blood and Separating the Serum.* This is a simple procedure if a properly blown Wright's tube is used in the manner herewith illustrated (Fig. 7). Failure to obtain a steady flow of blood into the body of the tube may be due to deficient and intermittent bleeding from the puncture, or to an improperly constructed tube. The first source of trouble is removable by making a puncture sufficiently large and deep to ensure a rapid, continuous movement of the blood column through the tube's capillarity, the end of which must be kept immersed in the blood until the tube is sufficiently filled, that is, to about two-thirds its capacity. In consequence of these precautions the capillarity does not become blocked by partly formed clots, as is the case when the blood oozes from the wound in single drops, the collection of which halts the onward movement of the blood as the tube is applied to each drop, thereby favoring more or less drying, coagulation, and interruption

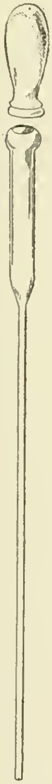

FIG. 4.—Mixing pipette.

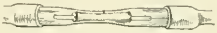

FIG. 5.—Wright's throttle.

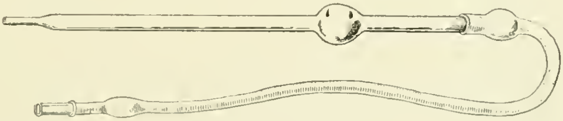

FIG. 6.—Suction pipette.

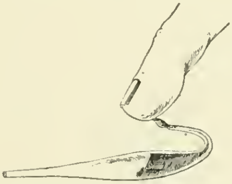

FIG. 7.—Method of collecting the blood with Wright's tube.

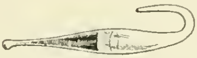

FIG. 8.—Serum and clot after centrifugalization.

of the column. The flow of the latter is materially hastened by gently heating the straight portion of the tube just before use. The employment of tubes conforming to the dimensions given above will eliminate perplexities referable to faulty apparatus. After collecting the blood the extremity of the straight portion of the tube is sealed in a flame and allowed to cool, after which it is grasped by its curved arm and shaken, as one shakes down the mercury column of a clinical thermometer, in order to drive the blood into the body and the tapering end. The tip of the curved arm is then sealed, to prevent evaporation, and the tube placed, straight end downward, in the centrifuge shield and centrifugalized, to separate the clot and the serum (Fig. 8). The latter is removed with a pipette at the time of the test, the wall of the tube having been scored with a file and broken.

II. *Preparing the Bacterial Emulsion.* About 10 or 15 c.c. of normal salt solution is poured into a culture tube containing a twenty-four-hour-old growth of Staphylococcus pyogenes aureus on an agar slant. The resulting emulsion is thoroughly shaken, transferred to another tube, and sedimented in a centrifuge. The upper, almost transparent, layer of the centrifugalized emulsion is pipetted off for use in the tests, since the lower opaque zone contains innumerable bacteria, many of them adhering in large, dense clumps, and therefore is unsuitable for use in the final enumeration. The proper density of the emulsion is best determined by experience, which will show that one which allows the phagocytosis of six or eight cocci by each leukocyte is easiest to work with; such an emulsion of Staphylococcus aureus should have but a delicately opaque appearance. MacFarland's nephelometer[11] may be tried as an aid in judging densities of bacterial emulsions.

III. *Obtaining the Washed Leukocytes.* At least ten drops of blood are collected and mixed in a test tube containing 2 or 3 c.c. of warm (37° C.) normal salt solution to which 1.5 per cent. of sodium citrate is added to prevent coagulation. It is important to use a solution of this percentage, which is three times stronger than originally advised by Wright and his students. A 0.5 per cent. solution of sodium citrate cannot be relied upon invariably to prevent clotting, which accident, of course, demands a repetition of this somewhat tedious step. The mixture of citrated blood is then centrifugalized until a compact corpuscular sediment, overlaid by clear citrate solution is thrown down, this sediment consisting of a deep red layer of erythrocytes, overlaid by a thin white or grayish film of leukocytes. With a suction pipette the supernatant fluid is removed down to the leukocyte film, which under no circumstance is to be disturbed. This accomplished, the layer of leukocytes and also the upper part of the erythrocyte sediment

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[11] Proc. Path. Soc. Phila., 1906, ix, 65.

(say to a depth of 5 mm.) are sucked up in another pipette (Fig. 9) and blown into a small test tube, in which they are well mixed by alternate suction and expulsion with the pipette. This upper layer of erythrocytes, whose presence in the field of the stained specimen is not undesirable, also contains many leukocytes. The mixture of leukocytes and erythrocytes thus secured, known as the "leukocyte emulsion," contains a considerable quantity of the sodium citrate of the original citrated salt solution in which the blood was collected, and which it is possibly advisable to remove. To do this the citrated salt solution is pipetted off and replaced by an equal volume of normal salt solution, which is thoroughly mixed with the leukocyte emulsion so that it effectually permeates it. The mixture is then sedimented in a centrifuge, after which the first salt solution is removed with a pipette, substituted by a fresh one, and the original mixing and sedimentation repeated. The removal of this second salt solution leaves a sediment of "washed leukocytes" practically free from the anti-coagulant salt and ready for admixture with the units of bacteria and serum, in the procedure of incubation, next in order.

Having found in our late work that sodium citrate has no influence whatever on the process of phagocytosis, we now dispense with this step of washing the leukocyte emulsion, thus appreciably simplifying the technique of the test.

It is in the foregoing stage of the test that most of the mistakes and perplexities crop out, from failure to secure enough leukocytes to enable one to make an accurate enumeration of the phagocytes and their engulfed bacteria in the stained film. This failure is referable to missteps in technique relating to the primary collection of the blood, to inaccurate pipetting, and to unavoidable adhesion of the leukocytes to the inner walls of the test tube and suction tube. In collecting the blood from which the leukocyte emulsion is prepared, at least ten large drops should be allowed to fall directly from the puncture into the citrated salt solution within the test tube, the blood not being permitted to trickle down the inner wall of the tube, to which the leukocytes tend to adhere with extraordinary tenacity. To this accident we have traced several failures to find sufficient leukocytes in the stained film, notwithstanding both macroscopic and microscopic evidence of an abundance of these cells in the sediment pipetted from the layer of the leukocyte emulsion, and in spite of an otherwise adequate technique. Fewer than ten good-sized drops of blood are insufficient to yield a leukocyte film of easily workable size, for with less than this amount of blood the sediment of leukocytes is so scanty that the number of these cells which survive subsequent manipulation is too small to be taken as a basis for an accurate calculation. Inaccuracy in pipetting accounts for numerous failures, but this is a fault to be remedied only by the acquisition of a perfect technique. It need scarcely be noted, save as a warning to the unskilled, that all the leukocytes may be inadvertently sucked up and discarded with the citrated salt solution during its removal in the step preliminary to washing the leukocytes, or that they may similarly disappear in the latter stage of the test. A test may be spoiled by neglecting to remove *all* the citrate or the salt solution above the layer of leukocytes, for in such an event the leukocyte unit will be found to consist of little else than a highly diluted collection of erythrocytes, the leukocytes being too few to be taken as the basis of a count.

IV. *Measuring, Mixing, and Incubating the Units.* A mark is made on the capillary stem of the mixing pipette about 4 cm. from its tip, the capacity of the tube between these two points being taken as the unit of volume for the patient's serum, the washed leukocytes, and the bacterial emulsion. Having placed these in their rack in the order illustrated (Fig. 1), the rubber nipple of the pipette is compressed and its point plunged into the bacterial emulsion which, by very cautiously relaxing the pressure on the nipple, is slowly sucked up to the exact level of the unit mark; the point of the pipette is then removed from the emulsion, wiped dry with a bit of cotton, and, by again carefully releasing the pressure, a small column of air is aspirated. This accomplished, a unit of washed leukocytes is similarly sucked into the pipette, then a second column of air, and finally a unit of the serum to be tested, the tube now containing equal units of bacterial emulsion, washed leukocytes, and the patient's serum, each separated by a partition of air (Fig. 10). These are now mixed by sucking them up into the expanded portion of the pipette, where they are thoroughly agitated and commingled by alternately squeezing and relaxing the pressure on the nipple, which should be manipulated with the greatest delicacy, in order to avoid sudden oscillations of the liquid and its accidental escape into the "nipple end" of the tube. When the admixture is completed the point of the pipette is sealed in a flame, after which the nipple may be removed without risk of disturbing the column of contained fluid (Fig. 11). This manner of mixing the units is preferable to mixing them according to the original technique, by alternately expelling the contents into a watch crystal and sucking them up the tube again. No better mixture is obtained by this older method, by the employment of which many leukocytes are lost[12] and myriads of small air bubbles are prone to form in the liquid, despite one's care to prevent such accidents. When sealed, the pipette is placed in an incubator, where it is kept for exactly fifteen minutes at a temperature of 37° C. A second pipette is filled in an identical manner, with equal units of the same bacterial emulsion, washed leukocytes, and the control serum from a presumably healthy person, and incubated in the same way.

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[12] If the contents of a pipette are blown out into a watch glass, numerous leukocytes adhere to its surface, and in consequence the mixture subsequently aspirated into the pipette and incubated is very deficient in leukocytes—perhaps so decidedly so as to preclude the possibility of making the final phagocyte count.

V. *Preparing and Examining the Stained Films.* After incubation the straight stem of each pipette is notched with a file and broken off, while to the other end is fitted a rubber nipple; the contents of the pipettes are then blown out upon separate glass slides or crystals and each thoroughly mixed with a platinum loop, so as to distribute the cells throughout the fluids. Films prepared from these specimens, either with cover-glasses or with slides, according to one's preference, may be stained with any good double stain, the subsequent examination being made with a $\frac{1}{12}$ inch oil-immersion objective (Fig. 12). In our work in the Jefferson laboratory Wright's modification of Leishman's stain has given the clearest differentiation of the bacteria and their cellular assailants.

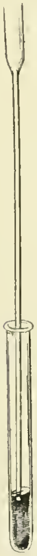

Fig. 9.—Method of aspirating the washed leukocytes.

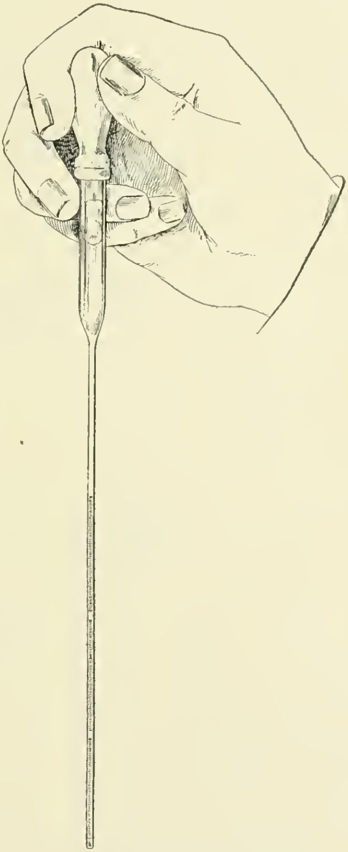

Fig. 10.—Mixing pipette containing the units of bacteria, washed leukocytes, and serum.

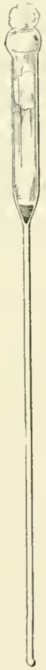

Fig. 11.—Mixing pipette containing the mixed units ready for incubation.

The final calculation of the opsonic index is based upon the enumeration of the bacteria found within at least 50 consecutive polynuclear neutrophiles in a film made of the specimen from the patient and in one from the control fluid. In making this estimate the bacteria simply clinging to the peripheries of the cells (not enclosed by them) were not included. Such groups of bacteria, however, were noted and recorded as "attached bacteria," for their appearance is most suggestive of an early phase of phagocytosis on the part of the leukocytes to which they adhere. The index of these extracellular bacteria is being calculated in our second series of diabetic cases, now under investigation. The two totals of phagocyted bacteria having been determined, that of the patient's specimen is divided by that of the control's, to express the opsonic index of the blood under examination. Thus if, say, 200 staphylococci were found in 50 of the patient's phagocytes and 400 in the same number of the control cells, the index for this ratio would read 0.5, or 200 to 400. It is a good plan also to calculate the percentage of phagocytic polynuclears, as advised by Simon,[13] thus obtaining an index comparable with and controlling the opsonic figure.

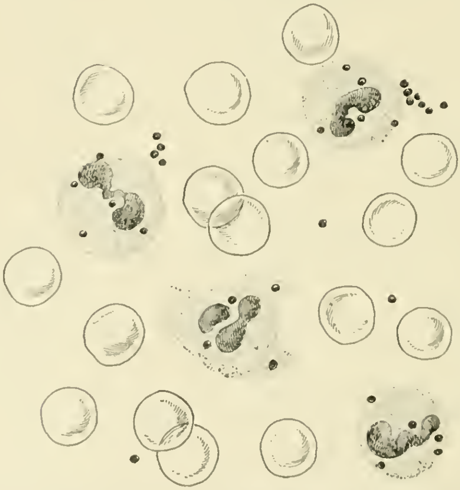

FIG. 12. Microscopic field of a specimen prepared from the incubated units. Note the staphylococci ingested by and adherent to the phagocytes; the latter are not included in the enumeration upon which the calculation of the opsonic index is based.

It will be noted that the polynuclear neutrophiles are the only variety of leukocytes taken into account in the foregoing enumeration, the number of phagocytic large mononuclear cells is so insignificant that they may be safely disregarded: only twice, during some fifty-odd examinations, have I noted phagocytosis of staphylococci by this form of the leukocyte, and in neither instance did the number of cells thus acting exceed three. It is possible, however, that with other bacteria (such for example, as the bacillus of leprosy) there may be quite a decided selective affinity on the part of these cells. Many of the eosinophiles were fragmented, clouds of granules being seen about the degenerate cell nuclei, and frequently lying in close contact with the bacteria free in the plasma. At first glance this reminds one of Kanthack and Hardy's description of the eosinophiles in lymph inoculated with anthrax bacilli, in which experiment it was found that the bacteria were primarily attracted by the eosinophiles, which, coming in contact with the germs, discharge their granules about them, with apparently crippling effect, since the bacilli soon show degenerative changes and later become the prey of the phagocytic large hyaline cells. It is, of course, far-fetched to attempt to distinguish any such phenomenon in the opsonin specimen, though the presence of free eosinophile granules near unengulfed bacteria in these films suggests more than a merely traumatic fracture of this peculiarly fragile type of cells, with the accidental grouping of their escaped granules in the neighborhood of the staphylococci.

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[13] Jour. Exper. Med., 1906, viii, 651.

In summing up the clinical value of the opsonin test, one may regard it as useful in two distinct ways: as a diagnostic agent, and as a means of gauging the effect of the vaccination method of treating certain infectious diseases. As a diagnostic agent the test should not, of course, be exploited in diabetes; nor, indeed is an additional aid of this sort necessary in this condition. A diabetic's opsonic index, however, gives a clear idea of the patient's resisting powers, of his susceptibility to secondary infection, and of the gravity of the inroads made by the disease. On the contrary, the opsonin test appears to be of considerable utility in the recognition of early tuberculosis, for in this infection the index to the tubercle bacillus is found to be either very low or excessively high; the former suggests at least a predisposition to the disease, while the latter points to an actual infection against which the subject's resisting powers are raised in defense. In an obscure case, an index to the tubercle bacillus ranging between 0.6 and 1.3 is a significant corroboration of otherwise puzzling symptoms. In still another manner is the opsonic index helpful in detecting such cases. The index of a normal person who has been injected with a small dose of tuberculin R. falls moderately for a period of about two days, after which it rises somewhat above normal, and finally returns to the original figure. In a tuberculous subject a similar injection is followed by a much more decided drop in the index, which remains subnormal for a week or so, and does not regain its original height for a fortnight or more. The fact that tuberculous effusions show a lower opsonic content than the subject's blood serum may also prove of diagnostic utility, as, for example, in determining the character of pleural or peritoneal exudates.

The most practical value of the opsonin test lies in its application to the vaccination method of treating bacterial infections, for in this class of diseases both the proper dosage of the vaccine and its effect upon the patient's powers of reaction are determined by the behavior of the subject's opsonic index during the period of treatment. Of this we are convinced, from personal knowledge, notwithstanding the criticisms which have recently been aimed at the test. This method of treatment, so ably devised by Wright and his pupils, is based upon the fact that the opsonic powers of the blood can be increased by the injection into the human body of vaccines, the essential steps being as follows: (1) Isolation of a pure culture of the bacterium responsible for the infection; (2) estimation of the patient's opsonic index toward the microörganism thus recovered; and (3) inoculation of the patient with suitable doses of vaccine prepared from this microörganism, in case the index be found subnormal. By this method of treatment it is possible, in many infections, to raise low opsonic indices to constant, high levels, and, in consequence of the habitual, active phagocytosis thus excited, to ensure disposal of the invading bacteria, and, hence, the recovery of the patient. That this may be done is shown by the numerous reports made during the past year, especially by those of the British school, whose treatment of acne, furunculosis, and abscess by staphylococcic vaccine, of malignant endocarditis by streptococcic vaccine, and of tuberculous diseases by tuberculin gives promise of even more favorable results in the future.

CHRONIC INTESTINAL AUTO-INTOXICATION.

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Intestinal auto-intoxication may be defined as that condition in which toxins formed in the intestines are absorbed by the organism in which they are produced. The use of this term has been objected to and enterotoxismus, or intestinal toxemia, has been suggested in its stead. Strictly speaking only that process should be called auto-intoxication which is caused by the toxic bodies resulting from metabolism and not due to any exogenous cause, such as bacterial activity. For the infectious process the term auto-intoxication is certainly inappropriate, but for the others Albu claims that they can be looked upon as autochthonic, in that they are caused by saprophytes which act only within the organism under special conditions. For the present, at least, it is impossible to determine which toxic bodies are the result of metabolism alone or the combined result of various causes. Moreover, the term intestinal intoxication is used by the French, the Italians, the Germans, and by ourselves, and it would seem that when the time comes that we can draw distinct lines between the various forms of intestinal intoxication it will be time to add new terms to our complicated terminology.

The whole subject was first brought to the general attention of the profession by Bouchard in 1887, and while much has been discarded which he claimed, yet much of the impetus given to the study of this subject is clearly due to the publication of his fascinating little book.[1] . Unfortunately the most important and characteristic of his symptoms, the toxicity of the urine, was not verified by subsequent observers (Albu, Forchheimer, and Stewart); so that, while such a clinical entity as chronic intestinal auto-intoxication was accepted, other proofs had to be found for its existence.

From a chemical point of view chronic intestinal auto-intoxication is due principally, if not entirely, to the intestinal changes in albumin and in nuclein. The changes in these two substances are the result of the activity of enzymes or of bacteria. The substances produced by enzymes are albumoses from albumin and the xanthin bases and uric acid from nuclein. In both instances pancreatic digestion is the principal cause. The action of bacteria manifests itself only upon albumin, as far as we know, in the form of pancreatic putridity and the bacterial processes which go on in the colon. The substances which result from this process and which interest us are phenol and indol, Brieger having shown that the rest of the bodies formed during putridity of albumin are little toxic. It has been shown that some of the albumoses formed during digestion are toxic, that is, when injected into lower animals. Of the purin bodies xanthin, hypoxanthin, paraxanthin, hetroxanthin, and adenin have also been shown to be toxic. Uric acid is toxic in a certain sense only; when it is injected into animals in large quantities no toxic symptoms are produced, but local symptoms may result under favorable circumstances. Both phenol and indol are toxic; in the lower animals indol produces decided symptoms (Herter); as the production of phenol and indol usually go hand in hand, the determination of one is sufficient for clinical purposes in order to determine the amount of putridity in the intestine.

As all processes due to enzymes are of relatively short duration, we are justified in assuming that all changes due to enzymes are completed in the small intestine, and this may also be the case in connection with uric acid and the xanthin bodies. As far as putridity goes, under normal circumstances little or none is found in the small intestine; indeed, Kohlbrügge says that the empty small intestine may not contain any bacteria. In the lower part of the ileum and in the colon, however, we find a large number of bacteria, and here is found the place where putrefactive changes take place. Not only the mere presence of bacteria, but the comparative lack of motion, therefore the greater the length of time in which these bacteria can act upon albuminous substances, makes it reasonably certain that putrefactive changes are confined to the colon under normal conditions. In the human being it does not take longer than a few hours (Krehl) for the small intestine to empty itself into the colon, so that we find everything which has not been absorbed by this time in the colon, including the metabolic products of nuclein, the xanthin bases. If for one reason or another the colon is not emptied sufficiently, a greater chance both for formation and for absorption of toxic bodies is given than when such a condition does not exist. From the intestines these various substances are absorbed and are sooner or later found in the blood, unless they are converted into harmless combinations. Those substances that are absorbed by the blood enter the liver and are subjected to oxidation, the so-called "filtering function" of the liver. Those that find their way through the lacteals are bound or changed by the lymphatic nodes through which they must pass.

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[1] Lecons sur les auto-intoxications dans les maladies.

As a result, a number of these toxic bodies are then rendered inert; some lose a part of their toxicity, others may pass through unchanged. In following the course of the circulation some of these bodies are excreted by the lungs, but relatively few, and then enter the left heart, going eventually to the kidneys, where many leave the body. Herter has shown that the cells of the liver, the kidneys, the muscle, the blood, and the brain have a decided effect, in the order mentioned as to quantity, upon a change in phenol and indol; so that the toxicity is being reduced all the time. During the circulation arterial blood containing toxins may be distributed to many parts and organs, the mucous membranes, the central nervous system especially, and may be eliminated there, producing symptoms or lesions. After the blood has come back to the right heart it has then been changed, as to these toxic bodies, depending upon the activity of the liver, the kidneys, the lungs, the muscle, the blood—probably by all the cells in the body. Two eventualities arise: either the toxic substances have all been removed, or this has not been accomplished. The latter is due either to a relative or an absolute insufficiency of the toxin-destroying organs or cells; relative in that the amount of toxin is too great; absolute in that for some reason the organs or cells do not perform their normal function. But after a time it does not remain a question of function alone; organic changes take place, either on account of increased functional activity, as upon elastic tissue, or because of irritation, as by the purin bodies, Gaucher, Crofton, Kolisch, Minkowski showing that they produced actual lesions in the kidneys.

For practical purposes the amount of toxic bodies formed depends upon the nature of the food, upon the time it remains in the colon, and the character of the processes within the intestine. Except in abnormal conditions the processes are the same, provided the other two factors are unchanged, for within certain limits the bacteria are the same. Under abnormal conditions the flora may be changed and then we may have peculiar auto-intoxicative bodies formed of which we know very little. Under all circumstances it is important to consider the time which is afforded the bacteria to act upon the contents of the colon, and the slow peristaltic function of the large intestine increases the activity of these bacteria in that they are not disturbed by motion. The character of the food limits the quantity and the quality of changes, but more or less putridity will always be found as the result of normal metabolism.

Turning to the practical aspect I desire to call attention to an analysis of 77 cases examined by ordinary clinical methods in which, as I believe, intestinal auto-intoxication was present. All the patients were observed in private practice. Children are excluded because the clinical picture in them differs very much from that in the adult. Of the 77 there were 37 males and 40 females, 43 patients being between thirty and fifty years of age, 48 having good habits, 12 being overworked, 10 irregular habits, 3 alcoholics, and 4 ex-cessive smokers. Their occupation was very varied Of the women, 31 were housewives; of the men, 16 were merchants; the professions were represented by 2 physicians and 2 lawyers. There were 3 farmers, 7 salesmen, and 1 broker. As all belonged to the so-called better classes whatever effect occupation may have had is not shown in this collection of cases. I have analyzed the chief complaints of these patients and find that they were general in 12 cases; 18 had some form of stomach trouble, 12 had intestinal and 52 had nervous troubles, 4 genito-urinary symptoms, 4 skin troubles, 17 some trouble with the locomotor apparatus, and 7 troubles with nose, eyes, or ears. Among the general troubles 8 complained of loss in weight, 3 of general debility, only 2 complained of their mouths and they had been sent by dentists for general treatment. Among those having gastro-intestinal troubles 2 complained of bilious attacks, 6 of constipation, 3 of alternate constipation and diarrhœa, and 2 of flatulence. The fact that only 11 patients should have complained of intestinal symptoms is one that will be referred to later. The great number of principal complaints were referred to the nervous symptoms, and here we find 7 cases of migraine, 17 with headaches, 6 with neuralgias, and 6 with vertigo.

The general appearance was good in 54 patients, not good in 21, and 10 were obese. In 14 of those noted as not good, the expression anemic was used, the other 7 were badly nourished, 1 having a cachectic appearance due to prolonged suffering from migraine plus morphine.

In the special examination I note only the salient points, as it would lead us too far to go into more minute detail. In the mouth we find Riggs' disease in 66 cases, normal mouth in 8, and no teeth in 3, therefore no Riggs' disease. Riggs' disease is called by various names: pyorrhea alveolaris, gingivitis interstitialis or parenchymatosa, stomatitis ulcerosa chronica. In a former publication upon the subject I suggested the latter name and divided the disease into three stages: (1) In which there is only the necrotic seam at the margin of the gum; (2) in which there is denudation of the teeth; and (3) in which there is pus infection. There were 21 in the first stage, 40 in the second, and 5 in the third. The fact that there were only 5 in the third stage is readily explained in that, as a rule, patients in private practice take good care of their teeth and certainly keep their mouths as aseptic as possible. As for the frequency of this manifestation in the mouth in chronic intestinal auto-intoxication, if we are at all upon the right track as to our understanding of the process in the intestine the existence of such large numbers of cases of Riggs' disease is almost a logical consequence. We have seen how the purin bodies are found in the intestine; we know that they produce necrosis of tissue; we have necrosis of tissue in Riggs' disease and in a large percentage of cases we find uric acid in scrapings from the teeth—so often has this been found to be the case that this form of disease has been called gout of the teeth. That this is the case I am prepared to testify as the result of a large number of examinations of scrapings from the teeth, the so-called blood calculi, in which I have always found uric acid. On the other hand, I should not like to be understood as saying that only the purin bodies, especially uric acid, may produce Riggs' disease.

Of the 77 patients 54 had stomach symptoms, 23 had none; 27 had dyspepsia, 19 had hyperchlorhydria, 1 achlorhydria, and 4 had other gastric neuroses, sensory or motor. In 7 patients there was nausea or vomiting and 1 had gastroptosis. Any explanation for the frequency of stomach symptoms in connection with chronic intestinal auto-intoxication must be purely hypothetical, as very little has been done to demonstrate which bodies are eliminated by the stomach; certainly nothing has been done, as far as I know, in connection with the various substances which interest us. This we know, that certain substances when introduced into the circulation are eliminated by the gastro-intestinal tract: thus the cholera toxin is formed in the intestine and is partly eliminated in the stomach from which it is removed by vomiting as Alt has shown. It is possible that the various substances formed in the colon may be eliminated into the stomach and there produce either functional or organic disturbance. But it is also possible that the nervous disturbance which exists to a great degree, as has been shown in intestinal auto-intoxication, may be the cause. Finally, the fact must not be lost sight of, that gastric disturbances, provided there be diminished HCl secretion plus deficient motility, may increase intestinal putridity. (Krehl.)

When I came to question and examine the 77 patients I found that all except 4 presented evidences of some trouble in the bowels. Eighteen were regular in their evacuations, 4 irregular, 44 constipated, that is, not having a movement daily, 5 had diarrhœa, in 5 there was alternate constipation and diarrhœa; 3 suffered much from flatulence and colic, and 5 had enteritis mucosa. That so many were found constipated is not an uncommon experience, for unless questioned about it many patients are apt to be silent on the subject. In examining the patients the history of 10 cases of appendicitis was elicited, 4 having had severe attacks from five to twenty years ago, 2 having the relapsing form, and 4 chronic appendicitis; 5 of these cases have since been operated upon and some of the conditions to be described were verified in them. The constipation which was present in them undoubtedly produced the symptoms of auto-intoxication, as these have disappeared since the operation of appendectomy. The alterations due to appendicitis seem to produce changes in the cecum which are followed by lack of peristalsis. One patient had a dilated colon, in 3 there was Glénard's disease.

In examining the colon by percussion and by palpation it was found that one or another part of it was filled in all but 4 cases; most commonly it was at the sigmoid flexure and in the descending colon, next in frequency came the transverse colon, then the cecum and ascending colon, and finally the hepatic and splenic flexures, that is, without any other part being involved. As a rule, we find combinations of several parts sometimes, as in 13 of the cases the whole colon was filled up. The most reliable results are obtained, by percussion, when the patient is in the upright position; for palpation the patient should be upon the bed; not infrequently it is necessary to turn him from one side to another or to lower the trunk. In making this statement I am fully aware of the untrustworthy results obtained from the percussion of the intestines in general, but here we have a something within the intestines which produces dulness when lightly percussed and whose presence can be verified by palpation. It is not safe, however, to trust percussion or palpation for a final opinion until further observation of the patient has taken place.

In order to be sure of the condition the fecal material must be removed from the colon in one way or another. In many patients this is not easily accomplished, as the fecal material has remained in the colon for a long time. In those patients having diarrhœa the diarrhœic material has passed without disturbing the old fecal material. It may be well to state that old fecal material does not produce auto-intoxication, but the condition of the bowel which results from it does. That the observations recorded above, as to retention in the colon are correct can be verified by successive and continuous examinations. Many of the patients when relieved either give up the treatment or neglect themselves in other ways; when they come back for observation the retained material is found in the same place where it was first detected. After the bowel has been thoroughly cleaned out it is then either found empty, *i. e.*, no dulness on percussion and no resistance on palpation, or there is resistance showing that the bowel wall is changed. According to my experience an empty bowel cannot be felt, as first claimed by Glénard and repeated by Sahli. When the bowel can be felt in the majority of cases its wall is thickened; this thickening may be due to a spastic condition, as suggested by Osler, or to a lesion due to a chronic catarrhal condition and infiltration of the walls of the intestine. When, after the removal of the collected fecal masses has been accomplished, no thickening of the intestines is found after repeated examination, we are justified in assuming that the fecal masses accumulate in the same place because peristalsis does not take place there; therefore an atony of the intestines. If, on the other hand, the thickening is found, and this usually occurs at the sigmoid flexure, there is some change in the bowel wall. In the greatest number of cases no thickening is left; therefore, a process due to faulty innervation, or atrophy. When the thickening is left, therefore, I believe it to be due to an organic process, a fact which is more definitely proved by the results of treatment than in any other way.

Before leaving the abdominal organs I wish to add that 3 of the patients had enteroptosis and 1 a fibroid tumor of the uterus, in which cases the interference with peristalsis was purely mechanical. In only 3 patients was the liver found enlarged.

The routine examination of the urine showed some changes which are worthy of special attention. The specific gravity showed nothing of importance, one-half of the cases being between 1009 to 1022, the other half above 1022 to 1030. Albumin (heat and nitric acid contact tests) was found in 6 cases. Indican was found increased in 68 cases. Obermayer's test was used: strong HCl, sp. gr. 1.19, to which is added 4 grams of ferric chloride to the liter. This we have used as a comparative test, by the adoption of a color scale. Simon has shown that indicanuria goes hand in hand with phenoluria, so that indican can reasonably be looked upon as an index to the amount of putrefaction which goes on in the colon. All the recorded cases were on mixed diet and had no other conditions which would produce an increase in indican elimination. It will be interesting to compare the increase in indican with the symptoms and the results of the physical examination. Of the 44 cases which were constipated 39 presented increase in indican. As far as this observation goes, it is in direct refutation of the statement made first by Jaffé in 1877, and since repeated by every author whom I have been able to consult. The case which Jaffé reports is not one which can carry much weight with it in evidence of his assertion in that it was one of intestinal obstruction of the colon. Curiously enough in the same volume of Virchow's *Archiv* there appears another article on the detection of the conjugate sulphates in the urine by von Velden,[2] in which the analysis shows that in one case of constipation the indican was increased. Baumstark has reached the same conclusion.

In all the cases in which there was alternate diarrhœa and constipation the indican was increased whether examined during the period of diarrhœa or of constipation. All the cases of mucous colitis had increase of indican. In the 13 cases in which the whole colon was found filled we found, on an average, the largest quantities of indican. Next to this came the sigmoid flexure and then the descending colon.

Microscopically there was found in the urine oxalate of calcium in 33 cases, uric acid in 11 cases, amorphous urates in 5 cases. Red corpuscles were found in 18 cases; it is difficult to account for their presence, sometimes only a few corpuscles, sometimes many were found. The subject is now being investigated, but for our purpose it is of interest to note that in 11 of the 18 cases oxalate of lime was also found. Hyaline cylindroids were present in 8 cases, hyaline casts in 15 cases, fine granular casts in 10 cases, once combined with hyaline casts, and once epithelial casts were present. The hyaline casts were found with albumin in the urine in 3 cases. It is interesting here to consider Dreser's statement that in all cases in which indican is increased there is albuminuria, because it produces irritation of the uriniferous tubules after having been taken up by their epithelial cells. We have 68 cases of increase in indican and only 6 cases of albuminuria. As far as the presence of hyaline casts is concerned, out of 15 cases we find 10 in which there were present large quantities of indican, the other 5 representing moderate increase. Only 1 of the cases in which albuminuria and casts were found is a chronic nephritic.

One other condition requires especial notice in that it occurred in 28 cases. These patients very frequently volunteer the statement that they have to pass large quantities of urine at times and frequently and in the course of the same day this condition may change to an oliguria. Or the condition of polyuria persists and is then followed by an oliguria, the one alternating with the other. This condition of polyuria may be ascribed to nervous processes, which, as will be shown, are very commonly associated with auto-intoxication. But the polyuric urine is most commonly of low specific gravity and in it the greatest quantities of indican is found. The oliguric urine, on the other hand, is of higher specific gravity and contains less indican; it would seem, therefore, that the indican acts, when present in large quantities, as an irritant to the kidneys.

In the women 18 of the 40 were afflicted with some abnormal form of menstruation; 13 had dysmenorrhœa, 3 menorrhagia, 2 insufficient and irregular menstruation. In a large number of these cases the same cause which produced the menstrual changes may be considered as the cause of the intestinal auto-intoxication; displacements, ovarian inflammations, peri-uterine inflammations, etc., in that the normal function was mechanically interfered with. But there remains a number of cases in which the gynecologist finds nothing wrong. In these cases it would seem that some toxic body may be also eliminated by the mucous membrane of the uterus; that substances are eliminated here can be easily demonstrated by testing the cervical mucus for iodine when the patient takes iodide of potassium.

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[2] 1877, p. 343.

Nervous symptoms were present in 61 patients. Of these there were 6 genuine migraines. All of these had large quantities of indican, 4 had constipation, 1 loose passages, and the others alternate loose and constipated movements; 2 had appendical troubles, 1 had a dilated and filled cecum, 2 sigmoid flexure trouble, and 1 descending colon and sigmoid flexure thickening; 31 patients had headaches, all of them had indicanuria except 3. All of them except 4 had some change in the bowels. Neuralgias were present in 11 patients—2 were noted as neuritis; 4 had paresthesia; 14 were nervous and restless; 7 irritable and excitable, with or without other symptoms of neurasthenia; 7 had depressive symptoms, 3 phobias, 11 insomnia, 2 hypochondriasis, and 1 melancholia; 4 were hysterical. Vertigo was present in 11 patients.

In 58 patients some deviation from the normal was found in the cardiovascular apparatus. Neuroses were found in 17; 11 had myocardial conditions; 6 chronic myocarditis, 3 dilated hearts, 1 the heart of arteriosclerosis, and 1 obesity of the heart; 12 had valvular lesions, 4 aortic insufficiency, 5 mitral insufficiency, 1 mitral insufficiency and stenosis, and 1 aortic and mitral insufficiency. In 3 others there was heard a soft systolic aortic bruit and in 5 there was an accentuation of the second aortic sound. There is no doubt in my mind as to the connection between neuroses of the heart and chronic intestinal auto-intoxication. That chronic myocarditis is produced by toxemia is still a matter of discussion. For the valvular lesions we could only be justified in considering those due to an intestinal intoxication which are combined with arteriosclerosis, that is, those mentioned last. Eighteen patients had arteriosclerosis. The discussion of the relation of intestinal auto-intoxication to arteriosclerosis would lead us too far from our subject. That arteriosclerosis is produced by gout is admitted, that gout, in so far as parin bodies are concerned in its production, is the result of intestinal auto-intoxication may also be admitted. How this produces arteriosclerosis has not been demonstrated, as it has been done with adrenalin and nicotine.

In analyzing the symptoms on the part of locomotor apparatus we again come to the question of gout, but here in a more definite form. In 50 patients there were found symptoms or diseased conditions in the joints or muscles. Of these 19 had gouty joints, 24 had muscular rheumatism, 13 complained of rheumatic, muscular pains which were transitory. One patient had intermittent hydrarthrosis of the knee. Out of the 19 with gross manifestations of gout 15 had increased indican. The formation of uric acid, and its antecedents, in the intestine evidently depends upon some of the same conditions that favor the formation of indican, that is, the quality of the food and the time required until it leaves the colon. Of the 24 who had muscular rheumatism 20 had indicanuria, the average being 3.5 with a maximum of 5 in 1 case. Transitory pains, muscular or otherwise, represented 11 cases of indicanuria out of 13; 22 cases presented skin manifestations; 5 urticaria, 5 eczema, 3 furunculosis, 4 acne vulgaris, 1 angioneurotic œdema (feet, toes, face; red spots), 1 dermatomyositis, and 1 with erythemata. One of the patients had red axillary perspiration and in 1 there was herpes zoster. Of these urticaria, eczema, erythema, and acne vulgaris have been ascribed to intestinal auto-intoxication as a direct or predisposing cause.

Only 13 patients had respiratory troubles; of these 5 had tuberculosis, 2 bronchitis, 4 bronchitis and asthma, and in 2 there was also emphysema.

The blood, when it was examined, presented the ordinary picture of a chlorotic anemia, with the exception of 3 cases, which will require further study.

The number of cases in which the nose, the eyes, and the ears were affected, that is, the mucous membranes, is small, principally because no note was made of their presence or absence unless there were special reasons for doing so. There were 6 cases of tinnitus aurium and 3 cases of Ménière's disease in which chronic auto-intestinal intoxication was found. In addition there was a number of nose, pharynx, and eye conditions which require further study, but which also seemed to be connected with auto-intoxicative processes.

If we now try to get a composite clinical picture from what has been said, in order that the diagnosis of chronic intestinal auto-intoxication may be made, the following will have to be taken into consideration:

1. In the gastro-intestinal tract there are Riggs' disease, various forms of stomach troubles, change in functional activity of the colon, and demonstrable retention of feces due to one cause or another.

2. In a large percentage of the cases indican is increased in the urine, and a microscopic examination shows, again in a large percentage of cases, calcium oxalate (50 per cent.), uric acid and urates (25 per cent.), red corpuscles (about 30 per cent.). In 28 cases, about $33\frac{1}{3}$ per cent., the alternation of polyuria with oliguria was noted.

3. One-half of the females had menstrual trouble.

4. Next to Riggs' disease, nervous symptoms were present in the

largest number of patients. Of these 31 of the 77 patients had headaches.

5. In a large number (58 out of 77) cardiovascular changes were observed; over one-half were due to neuroses and to myocardial conditions.

6. Of the 77, 50 patients had locomotor symptoms, gouty joints, but especially muscular symptoms.

7. Twenty-two patients had skin lesions.

From the combination made by various symptoms, the diagnosis of chronic intestinal auto-intoxication should be easily made, and, as a matter of fact, this is the case in practice, as is shown when this diagnosis is followed by the logical therapeutic pressures.

But it is much more difficult to determine how much importance the intoxication may be in an individual case—whether it produces all the symptoms, whether it is the cause or the result of other conditions, whether it is the direct or the predisposing cause. It is a great temptation to make this diagnosis, as it is to make all diagnoses that can be so generally applied as this one. Therefore the utmost self-criticism should be used before chronic intestinal auto-intoxication should be looked upon as the final diagnosis. In these cases which I have collected this diagnosis was made only ten times. In all the other cases the auto-intoxication was looked upon simply as a contributory cause, present and requiring treatment possibly, but not the principal disease.

THE AUSCULTATORY AND PERCUSSION FINDINGS OF THE HEART IN LATE CHILDHOOD.

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There is probably no period of life in which the heart findings during physical examination are more frequently misinterpreted and consequently misjudged than in late childhood. This seems to depend on two factors: (1) The comparatively large cardiac area; and (2) the frequency with which murmurs are heard over the heart at this time. With these points in view, a number of children, not patients, were examined. The results obtained in 100 of these, free from cardiovascular or renal disease, anemia, or abnormal abdominal conditions or distention, are given herewith.

Before presenting an analysis of the examinations an explanation of the plan adopted in calculating the cardiac outline seems necessary. Palpation and percussion were employed, and the lines of deep cardiac dulness were taken. These are more easily outlined in children than in adults, for reasons well known; namely, the thin chest wall, the thin layer of lung tissue overlying the heart, and the less independent vibration of the sternum and anterior thoracic wall during percussion. These factors also account, to some degree, for the larger cardiac area in children.

The upper and right borders were obtained in the erect, and thereafter in the recumbent position. The left border was similarly obtained when the apex beat was not distinctly evident or palpable. While the apex beat does not always represent the extreme left limit, it, in general, will give, I think, more accurate results than percussion.

The ribs and interspaces were regarded in estimating the upper border and the sternal and nipple lines, respectively, in calculating the right and left borders. While the nipple may vary in position, I think the mammillary line will prove quite as accurate as the lines that may be dropped from the clavicle, in determining the midpoint of which undoubtedly greater errors are possible. On the other hand, measurements of distances of cardiac area to the left from the median line were not employed, because this does not convey a practical conception of the subject. The children studied had passed the sixth year and were of varying ages up to puberty.

By percussion in the parasternal line the upper border was found in the erect position, as follows:

Location.	*Number of children.*
On the second rib	1
In the second interspace	4
On the third rib	65
In the third interspace	17
On the fourth rib	13

The right border, obtained on a level with the fourth rib and the fifth interspace, in the erect position, was found:

Location.	*Number of children.*
On the left sternal border in	3
In the mid-sternal line in	12
On the right border of sternum in	8
0.5 cm. to the right of the sternum in	13
1.0 cm. to the right of the sternum in	32
1.5 cm. to the right of the sternum in	18
2.0 cm. to the right of the sternum in	14

The dulness obtained to the right of the sternum is always slight, and is brought out only by light percussion. Heavy percussion may bring out dulness beyond the actual heart outline in a child (Sahli).

The apex beat in the erect position was located with regard to the nipple line, as follows:

Location.	*Number of children.*		
	Inside N.L.	*On N.L.*	*Outside N.L.*
In the fourth interspace	3		3
In the fourth interspace and behind the fifth rib	3		
In the fourth and fifth interspaces			1
Behind the fifth rib	4	4	2
Behind the fifth rib and in the fifth interspace	4		
In the fifth interspace	52	19	5
In the fifth interspace and behind the sixth rib	1		
Behind the sixth rib and in the sixth interspace			1

The upper border in the dorsal position was:

Location	*Number of children.*
Unchanged in	45
Elevated the width of an interspace	19
Elevated the width of a rib	11
Elevated the width of a rib and an interspace	5
Dulness noted as more pronounced in	3
Dropped one interspace in	3
No record in	14

The right border in recumbency was:

Location.	*Number of children.*
Unchanged	43
Diminished 0.5 cm.	13
Diminished 1 cm.	16
Diminished 1.5 cm.	5
Dulness noted less marked	10
No record	13

The apex in recumbency was:

Location.	*Number of children.*
Unchanged	57
Elevated from the fifth interspace to behind the fifth rib	13
Elevated behind the fifth rib and into the fourth interspace	4
Elevated from the fifth to the fourth interspace	4
Less distinct with position unchanged	8
Shifted to outside nipple line with horizontal position unchanged	7
No record	14

Symington, in discussing this question from an anatomical point of view, states that since the vertical extent of the heart in relation to the anterior chest wall is practically similar in children and adults, it naturally follows that the transverse diameter of the heart, as compared with that of the chest, is relatively greater in the former (in whom the characteristic feature of the thorax is its narrowness) than in the latter.

The average diameters of the thoraces of the children examined, taken in the upright position, were as follows:

Age	*Lateral diameter.*	*Anteroposterior diameter.*
7 years	6.500 inches.	5.625 inches.
8 "	7.125 "	5.650 "
9 "	7.530 "	6.053 "
11 "	8.060 "	6.150 "
12 "	8.600 "	6.620 "
13 "	8.480 "	6.500 "
14 "	8.700 "	66.500 "

Contrary to the observations of Gordon, who found a decrease in the anteroposterior diameter in the dorsal position, I found either a slight increase or no change in this diameter in recumbency, only occasionally noting a slight decrease. Mathes records similar results in his article on enteroptosis.

Gierke took measurements of the length of the heart from the apex to the middle of the right border (which latter was represented by a line drawn from the upper point of the heart in the middle line on a level with the second rib to its lower border to the right of the middle line), in addition to measurements of the lateral diameter of the chest. I quote only those examined over six years.

Age.	*Lateral diameter.*	*Heart's length.*	*Per cent. of heart's length of lateral diameter.*
7 years	14.00 cm.	9.0 cm.	64
8½ "	18.00 cm.	9.5 cm.	53
9 "	19.00 cm.	10.0 cm.	52
9¼ "	16.50 cm.	12.0 cm.	72
9½ "	18.25 cm.	9.5 cm.	52
9¾ "	19.00 cm.	10.0 cm.	52
10 "	15.00 cm.	11.0 cm.	73
10 "	20.00 cm.	12.0 cm.	60
10¼ "	17.00 cm.	9.0 cm.	53
10½ "	16.00 cm.	10.0 cm.	62
11½ "	18.50 cm.	12.0 cm.	66
12 "	16.75 cm.	10.0 cm.	59
12¾ "	20.00 cm.	10.0 cm.	50
13 "	19.00 cm.	10.5 cm.	55

Of the children examined by Gierke, it will be noted that the length of the heart was equal to the percentages given of the lateral diameter varying from 50 to 73 per cent. The measurements of heart length, however, are not parallel to the transverse diameter, the former being at an angle of about 25° to 30°. The above represent individual cases and not averages of a number of cases.

While a verification of my findings by the *x*-rays would be desirable, this, for evident reasons, was found impractical. I might mention, however, as a basis of comparison, the findings of Dusch and Steffen. Dusch states that the apex may be found inside, outside, or in the nipple line; that the left border extends slightly beyond the apex; that the right border projects a little to the right of the right sternal border; and that the upper border is found in the second interspace or behind the third rib. Steffen found the right border 1 to 3.75 cm. to the right of the middle line on a level with the fourth interspace and fifth rib.

It will be noted that if we combine the more definite parts of Dusch's statements with those of Steffen, they represent closely my findings.

To sum up the findings of the 100 children examined, it may be stated that the upper border percussed in the parasternal line is most frequently found on the third rib, less often in the second or third interspace; that the right border may extend 0.5 to 2 cm. to the right of the sternum, as indicated by a slight relative dulness only; and that the apex, while most frequently found in the fifth interspace inside the nipple line, may be found outside of it. It is seldom in the fourth interspace, in which event it is as often inside as outside of the nipple line.

It will be noted that in changing from the erect to the dorsal position no change is found in the level of the upper or right border in about 45 per cent. of the children, and that in many of the remainder a difference of 0.5 to 2 cm. was found either in elevation of the upper border, or diminishing of the right border, there being no parallelism in the changes in these borders in most of the children.

In a few children the upper border was lowered in the recumbent position. In regard to the position of the apex, in more than half it remained unchanged, though at times it became less distinct; in others it ascended the width of an interspace or a rib, or of both; and in a few instances, without change in horizontal level, it glided slightly to the left.

The differences in the cardiac area, as found in the erect and the recumbent positions, undoubtedly depend upon the two factors often cited: elevation of the diaphragm and gravity.

The importance of an accurate knowledge of the area of cardiac dulness in children might not be apparent were we to neglect to study at the same time the auscultatory findings. I have, therefore, attempted to tabulate some of the results:

Auscultation was carried out in the erect and in the recumbent position. Of 94 cases in whom the tones were noted the—

	Number of children.
First tone at the apex was louder standing in	59
First tone at the apex equally loud or louder in recumbency in	35
Second tone at the apex was louder than the first tone in erect position in	5
Standing.	
Pulmonic tone was louder than aortic in	65
Aortic tone was louder than pulmonic in	21
Pulmonic and aortic tones were equally loud in	8
Recumbent position.	
Pulmonic tone was louder than aortic in	77
Aortic tone was louder than pulmonic in	4
Pulmonic and aortic tones were equally loud in	13
The first tone appeared at times divided in	12
The second tone at the apex appeared divided in	1
The second pulmonic was divided in	12

ACCENTUATION OF SOUNDS.

Standing.		*Reclining.*	
Pulmonic.	*Aortic.*	*Pulmonic.*	*Aortic.*
4	2	5	0

Murmurs in the cardiac area were heard in 64 cases. Of this number in only 18 were they heard in the erect position, leaving 46 in whom they were audible only in recumbency. Murmurs heard in the erect persisted in the recumbent position.

In the 18 children referred to the murmurs were always systolic in time and localized as follows:

Location.	*Number of children.*
At the apex in	2
At the apex and the base in	6
At the apex and the pulmonic area in	2
In the pulmonic area only in	6
In the pulmonic and aortic areas in	2

The localization of the murmurs which were all systolic in the 64 children in whom they were heard in recumbency was as follows:

Location.	*Number of children.*
Over the apex only in	3
Over the apex and pulmonic areas in	17
Over the apex and pulmonic and aortic areas in	25
Over the pulmonic and aortic areas in	8
Over the pulmonic area in	7
Over the apex and aortic areas in	2
Over the aortic area in	2

The murmurs were much influenced by position: those heard over the apex and the pulmonic area were usually intensified on placing the child on the left side, and were well heard along the left limit of the heart; when the murmur was heard only over the apex or the pulmonic area it often appeared at either point when not previously heard. Occasionally the murmur was little affected and was even diminished in the left position.

On the other hand, in the right lateral position, the murmur sometimes appeared along the right side of the sternum and was louder over the aortic area than in the dorsal position; in a few children it occurred for the first time over the aortic area under these conditions.

In one child, after distention of the stomach by gas, a systolic murmur appeared over the base of the heart. In a few instances the murmur seemed intensified after inflating the stomach.

Steffen mentions a case in which a systolic murmur was heard over the heart on pressing an enlarged liver upward. He attributed it to direct compression of the heart.

The murmurs varied considerably, at times, in loudness under the auscultating ear, were sometimes inconstant, and frequently disappeared at the height of inspiration or were greatly diminished in intensity, being usually heard best during the expiratory phase; they were also heard well during the ordinary respiratory excursions. They were seldom heard much beyond the cardiac area, were usually loudest over the pulmonic area, but not infrequently so over the apex. They are not loud murmurs, as a rule, and are sometimes so low as to be easily overlooked.

From the mentioned characteristics of these murmurs it is evident that they do not conduct themselves as do organic murmurs, which are not markedly influenced by position or the respiratory movements. We must, I think, look upon them as accidental murmurs, and accidental murmurs of striking similarity, often induced and almost always influenced by position and respiration, all of which suggest that they are dependent on the relation of the heart to its environment, that is, the lungs, or *vice versa;* in other words, we are dealing with extracardiac, paracardiac, or cardiopulmonary murmurs, occurring in the systolic phase of the heart cycle.

It may be assuming considerable to state that all these murmurs were cardiopulmonary, but in all those instances in which position and respiratory movements exerted such a marked effect (and these constituted the greatest number) such an assumption seems permissible.

In view of the association made by some between the presence of accidental murmurs and assumed low arterial tension, I wish to state that the average blood pressure in these children, taken with the Riva-Rocci apparatus (narrow armlet), was 119 mm. Hg., which is one point higher than that given by Hayashi for 108 children of similar ages examined with the same instrument. In no instance was the blood pressure below 100.

The cause of these murmurs appears to be, as has been pointed out, the varying tension exerted by the heart on the overlying and adjoining lung tissue which, when diminished during the systole of the heart, permits of air entering or being sucked into previously compressed vesicles, thus giving rise to a cardiopulmonary gush in the systolic phase of the heart cycle. In one or two instances the murmur seemed prolonged into diastole.

The increase in loudness of the murmurs often when the subject is placed in the left-sided position, and the appearance of the murmur to the right of the sternum, where it had not previously existed, when the child was placed in the right lateral position, is strong support of the above premises of their causation. Likewise, the fact that if the intrapulmonary tension is increased by deep breathing and holding of the breath, the murmur often disappears entirely, is equally convincing.

The greater frequency of these murmurs in the recumbent than in the upright position is apparently due to the ascension of the heart, as indicated in many instances by the elevation of the upper border and the apex, the result of which would be the pressing aside and above of overlapping lung tissue, producing conditions favorable for the development of cardiopulmonary murmurs. These conditions are not only favorable for the development of accidental murmurs, but are also favorable for better auscultation of organic murmurs, by a greater bearing of the valve area.

I have purposely avoided mentioning and commenting on the numerous theories that have been suggested in explanation of accidental murmurs, and have merely suggested the cardiopulmonary theory as a plausible one for the murmurs of late childhood, presenting the characteristics above stated. An explanation of the cause of the auscultatory phenomena of the heart heard in these children is of far less practical importance than their recognition and the knowledge of their significance or lack of significance.

The conditions found in late childhood with regard to the area of dulness and the auscultatory findings of the heart are such as may lead to considerable confusion in their interpretation, and undoubtedly constantly do, when they are not well understood. They have frequently been supposed to mean dilatation of the left ventricle, relative mitral insufficiency, strained ventricle, etc. A clear understanding of the conditions existing in children as to the area of dulness and auscultatory findings of normal hearts would probably avoid many such errors, because the presence of accidental murmurs (cardiopulmonary) in childhood is, I think, devoid of pathological significance.

The frequency of accidental murmurs in the children examined by Hamill and Le Boutillier[1] were practically the same as in those examined by me. The differentiation they draw, however, between late systolic pulmonary murmurs, which they incidentally refer to as an abnormal condition, and cardiopulmonary murmurs, appears to me a distinction without a difference. I consider it quite impractical to describe refined qualitative differences in murmurs, with the expectation that it will appear the same to other than the one who described them, for, after all, differentiation of systolic murmurs on this basis is not tenable. In other words, it is not practical to differentiate by the character of the murmur between accidental murmurs over the heart area, presumably dependent on different causes, nor is it practical always to differentiate between accidental and organic murmurs from the qualities of the murmur. This at once raises the question of the value of the systolic murmur in general as a diagnostic sign, whence an accurate knowledge of the normal heart becomes indispensable to a decision in children. The systolic murmur is only one of the several signs essential in the diagnosis of valvular lesions.

IMPROVED METHODS IN THE SUCCESSFUL OPERATIVE TREATMENT OF CLEFT PALATE.

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The advisability of plastic operations for the repair of congenital clefts of the palate was for a long time debatable—because successful closure of the gap and a fair restoration of normal speech were unusual. The rejection of osteoplastic operations and the adoption of ingeniously constructed obturators with a movable velum were earnestly advocated by some surgeons.

After a thoughtful experience with various forms of operation and with obturators, I have come to certain definite conclusions which may be worthy of consideration. I have been led to the presentation of these views by the recent publication of articles on the treatment of cleft palate, which show me that my experience and practise differ from those of some other American operators. In some essential points my opinions have been radically changed within the last few years; in others, I have reached conclusive views, where previously my mind was unsettled.

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[1] Amer. Jour. Med. Sci., 1906, cxxxiii, 55.

The view that operations on fissures of the palate should be delayed until the child has become two, three, or four years old is erroneous. It is better to operate when the infant is only a few days old, unless there be some very grave physical debility. In that event the operation may be delayed a few weeks, but such a delay is a misfortune. The time thus occupied in building up the infant's health may be profitably employed in digital compression, applied daily to the two halves of the upper jaw. Squeezing the separated segments of the hard palate together a few times every morning and evening will tend to lessen the breadth of the cleft and give the surgeon a better opportunity of obtaining a bony roof to the mouth by operation.

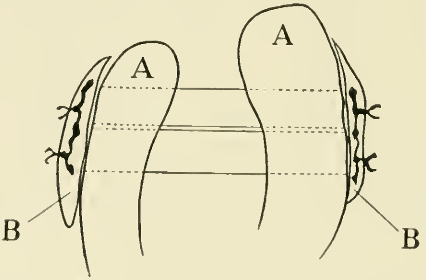

Fig. 1.—Diagram of Brophy's method of forcing and holding the two halves of the infantile cleft palate together by means of silver wires passed through the jaw above the alveolar process. By repeated pressure and gradual tightening of the wires, at the time of operation, the osseocartilaginous roof of the mouth is reconstructed. A, alveolus; B, lead plate. View from the mouth.

Better than this, however, is operation a day or two after birth. My belief is that the proper course, then, will usually be to adopt the method of Brophy, who, by means of long needles, carries strong wires through the two upper maxillary bones above the level of the palate (Fig. 1). The ends of these wires are passed through holes in leaden plates, placed on the external, or buccal, surface of the bones. After the two parts of the hard palate have been forced as near together as possible, by the surgeon's fingers, the adjoining ends of wire are twisted and the bones thus held in the new position.

This procedure causes the loss of but a few drops of blood, gives rise to little or no surgical shock or pain, and has the inestimable advantage of restoring wholly or in part the normal width of the defectively united roof of the mouth. Even if the two upper maxillary bones cannot be brought absolutely together much is gained by diminishing the width of the cleft in the upper jaw. If restoration of the oral roof is not done thus early the sucking and chewing of the babe and future child perhaps tend during the first years of life to increase the width of the palatal fissure. After the use of the wire tie-beams, as they may be called, for about eight weeks, mucoperiosteal flaps from the oral surface of the palate may be used to close any remaining gap in the hard palate, and the soft palate may be closed by freshening and suturing its edges, which now lie comparatively close together, or by using flaps of mucocellular tissue, as in Lane's operation. In narrow clefts the soft palate may be sutured at the time the wires are introduced to bring the two halves of the bony palate together (Fig. 2).

This operation seems to me to be a most valuable addition to our resources. I believe it to be philosophically surgical, since it has for its foundation the idea that the bony elements of the normal partition between nose and mouth should be compelled to aid in the restoration of function to those two cavities.

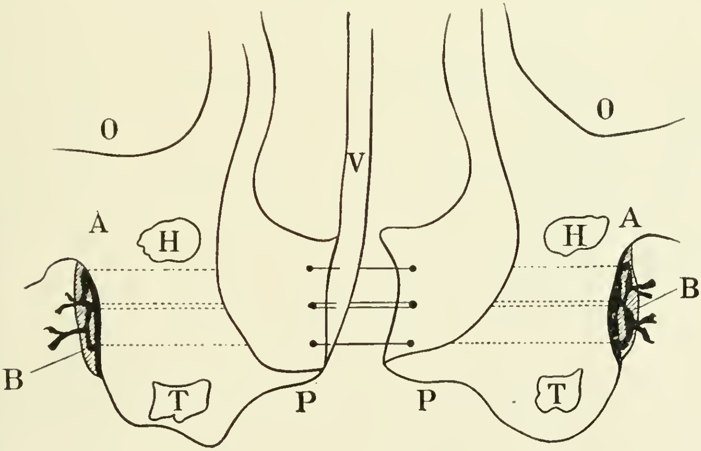

FIG. 2.—Diagram of a vertical view of Brophy's tie-beam method: A, jaw bone; P, palate; T, tooth; H, antrum; B, lead plate and wires; V, vomer; O, orbit.

The closing of any gap left, after all that is possible has been gained by the Brophy operation, I would usually effect by mucoperiosteal and mucocellular flaps made and sutured after the manner of Lane. This method is also to be employed, without a preliminary osteoplastic procedure, in patients in whom the operative treatment has been postponed until the child is over five or six months old, and in cases in which the cleft involves only the soft palate.

One should not be tempted to operate on the harelip, if this coexist, before treating by operation the palatal deficiency. It was formerly a common practise to correct the labial deformity early in infancy, and postpone the operation on the roof of the mouth for several years. This is an error. The great improvement in the physical appearance of the child resulting from the closure of the harelip is admitted. It is much more important, however, to close the roof of the mouth as soon after birth as possible, in order that the nasopharynx and accessory sinuses of the nose may be developed by the pressure of inspired air. The whole facial appearance of a growing child is changed by the direct and indirect influence of this conversion of a mouth breather into a nose breather. An additional result may be expected from closing the opening in the oronasal partition of the baby. It is the avoidance to a great extent at least of the catarrhal rhinitis and pharyngitis so common in cleft palate patients.

Closure of the lip does not effect these desirable improvements in the child's health and facial contour; and, if done before the palate operation, it is in a sense detrimental, because it interferes somewhat with the necessary uranoplastic manipulations by lessening the ease of approach to the operative field. The proper course, therefore, is to close the palate first and to operate upon the lip subsequently. The two operations at times may be done under the same anesthesia, or the labial operation may be delayed a couple of months.

When a cleft palate patient is over six months old it is difficult, if not impossible, to press the two separated upper jaw bones together, so as to close or diminish the intervening cleft. The cartilaginous character of the newborn baby's skeleton is lessened at that age by increasing deposition of inorganic salts. The exact age at which the bones become too rigid for successful displacement varies. In the early weeks of life they may be bent into the desired position without fracture occurring; later they may sustain a greenstick fracture through the body of the jaw above the malleolus. In some cases it is necessary to divide the two upper maxillary bones at this point by a horizontal section with a saw.

This increasing difficulty of forcing the bones toward the median line, so as to hold them together with wire tie-beams, is the chief reason for early operation. Brophy maintains that in most cases of cleft hard palate there is no real deficiency in the amount of osseo-cartilaginous material to form the roof of the mouth; but that the two halves have simply failed to unite and are spread apart. Hence the value of his method of forcing the palatal processes together and holding them in apposition until union is obtained. The importance of giving the muscles of phonation and the bones of the mouth and nose an early chance to develop and perform their physiological duty will be recognized by all. Delayed operative reconstruction of the palate means progressive atrophy of muscles and other tissues from disuse, and the development of abnormal relations and activities of the parts.

When the palatal segments cannot be brought together even after sawing the bodies of the bones, or when the true physiological time for this operation has passed, the surgeon should close the gap in the bony palate by means of flaps of mucoperiosteum cut and sutured as suggested by Lane (Fig. 3). I have had far better success by this method than by displacing two flaps toward the middle line, as is done in the Langenbeck and Fergusson operations.

Lane raises a large mucoperiosteal flap from the side of the fissure, where there is the larger expanse of palatal tissue. This flap is cut with its free convex border at, or actually upon, the alveolar process, and is left attached at the margin of the congenital cleft. On the opposite side a flap is raised, which is cut loose from the bone at the margin of the cleft and remains attached, for its blood supply, to the bone near the alveolus. The first flap is then turned over, laid across the fissure in the bone, and tucked under the flap raised on the opposite side of the palate. It is sutured in this everted position by two rows of delicate silk sutures inserted with very small curved needles (Fig. 4). The raw surface of the first flap faces the tongue and subsequently becomes cicatrized with a pseudomucous membrane.

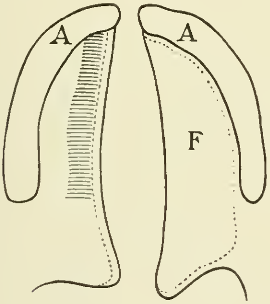

Fig. 3

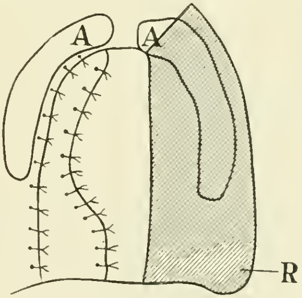

Fig. 4

Fig. 3.—Diagram of the roof of the mouth, showing incisions by Lane's method. A, alveolus; F, flap which is to be everted and tucked under the raised flap on the opposite side. The shaded portion indicates where the raised flap is to be separated from the hard palate.

Fig. 4.—Diagram showing the appearance after the flaps in Fig. 3 have been sutured: A, alveolus; R, raw surface left after the flap has been everted.

This method of utilizing the flaps has the advantage of covering the gap without much tension of the tissues. In addition the secretions from the nose have slight access to the suture lines and therefore the suture punctures are not likely to become infected. The raw surface of the large flap is constantly cleaned by the patient's tongue (Fig. 5).

When the operation is done before the child has developed molar teeth the first flap may be made of extreme width by cutting it from the top and outer side of the alveolar process as well as from the surface of the palate. It may even extend upon the cheek. I know of no other method of uranoplasty which makes available such an extent of well-nourished tissue.

About eight years ago I endeavored to close a palatal cleft by everting a mucoperiosteal flap from each side of the fissure and suturing them in the middle line. I did not know of Lane's method at that time. The cicatricial contraction of the periosteal surfaces made the flaps curl up toward the mouth and caused a failure, after I had thought successful closure of the gap was accomplished. Lane has solved this difficulty and made the plastic operation generally successful by everting only one flap and tucking its free edge under a raised flap on the opposite side of the congenital fissure. When I used the method of Langenbeck and Fergusson, I always entered upon the treatment of cleft palate with misgivings. Now, on the other hand, I am always glad to see such patients, whether they be poor or well-to-do, for I feel so much more sure of obtaining a good result.

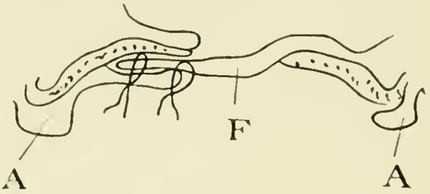

Fig. 5

Fig. 5.—Diagram to show in section the relation of the flaps after suturing in Lane's operation: A, alveolus; F, large flap which has been everted and tucked under small flap.

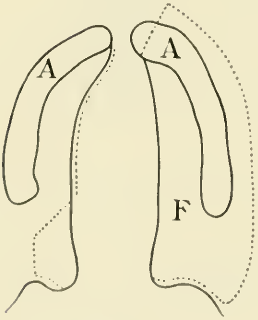

Fig. 6

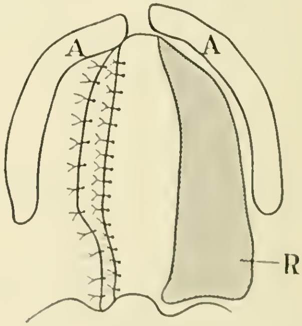

Fig. 7

Fig. 6.—Diagram to show incisions for flaps in Lane's method when the cleft is very wide, and the large flap must include the mucous membrane over the alveolus. The posterior part of the incision on the left side of the diagram is on the nasal surface.

Fig. 7.—Diagram to show appearance when in Fig. 6 the flaps have been sutured

It not infrequently happens that the entire palate cannot be repaired at the first operation; but subsequent minor operations, so common in all plastic work, finally complete the reconstruction (Figs. 6 and 7). With the early closure of a cleft palate made practically always possible and the operative treatment of harelip so satisfactory as it has now become, in skilled hands, surgery holds out to the unhappy parents of the deformed little ones a great deal of comfort.

I formerly used obturators, or artificial palates, occasionally, because of my fear of operative treatment being less satisfactory. These cannot be worn until the child has attained nearly the age of adolescence. Then the secondary atrophy of the nasopharynx and facial skeleton have become established and the speech defects confirmed. Mechanical appliances are, therefore, to be usually rejected in the consideration of the treatment of congenital clefts of the palate. They may at times be employed when the patient has been allowed to grow up without operative treatment in infancy; but they are even then probably a poor makeshift for reconstructive treatment by operation.

In all cases of this congenital anomaly systematic instruction in respiration and speech should be begun immediately after the palate has been repaired by operation. The child must dilate, as it were, the nasopharynx and accessory sinuses of the nose, and train the muscles of phonation, as well as the corresponding brain centres, to do their full physiological duty.

In conclusion, I state my opinions as follows: Cleft palate should be operated upon as soon as possible after birth. Accompanying harelip should not be treated until after the palate operation. When the operation is done during the first six months of life, Brophy's tie-beam method should usually be adopted.

After six months of age, the Lane flap method should generally be employed. At times it may be needed as a supplement to Brophy's method in younger patients. Brophy's method for these cases by means of silver-tension sutures and coaptation sutures with lead splints, or plates, applied to the inferior surface of palate flaps, is also very valuable.

Mechanical appliances to close the fissure are inferior to operative treatment, which has a low necessary mortality.

Instruction in speech is a necessary adjuvant for satisfactory results.

OBSTETRIC PARALYSIS.

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No better illustration of the rapid advance of medical knowledge and treatment within the last quarter of a century can be found than that offered by the history of obstetric paralysis. It was first brought to the notice of the medical profession by Duchenne in 1872. Yet even in 1886, Thorburn[1] could find but five articles on the subject. Since 1900, however, a certain number of cases have been described in the literature and a few articles have been written both on the etiology and on the surgical treatment. Although the condition appears to be a comparatively simple one pathologically, it is only of late that it has been understood. Many plausible theories have been formed, but it is only within the last few years that the visible pathological proof has been found and the evidence from experiments and dissections has been obtained.

For practical purposes we may divide obstetric paralysis into three forms: (1) The upper-arm type; (2) the lower-arm type; and (3) the two preceding combined, paralysis of the whole extremity. The lower-arm type is rare. Dr. Thomas[2] has recently described and discussed the cases of this character.

The common form of obstetric paralysis is the upper-arm type, corresponding in a general way pathologically to Erb's paralysis in the adult. This includes the large majority of cases of obstetric paralysis which are commonly seen, and I shall restrict myself to the consideration of this form. It was suggested by Erb that this type of paralysis is due to inhibition of conduction, whether by pressure, section, or destruction of the fifth and sixth cervical nerve roots, and he determined that faradization of these nerve roots irritated or caused contractions of the muscles paralyzed. Later, within the last ten years, operations performed on the brachial plexus in cases of Erb's paralysis in adults have confirmed the fact that these nerve roots are injured in this form of paralysis, and conversely have tended to prove that the injury of these nerve roots is the cause of this paralysis. The cause of the lower-arm type is injury of the seventh and eighth cervical and first dorsal nerve roots; when the whole limb is totally paralyzed, all the nerves, fifth, sixth, seventh, and eighth cervical and first dorsal, are affected.

Although the position of the lesion in obstetric paralysis had been assumed as a fact from the time of Duchenne, the cause of this lesion long remained obscure. Nearly all the earlier writers attributed it to pressure. Duchenne, followed by others, believed that direct pressure by the forceps on the nerves going to form the brachial plexus was the cause. Others asserted that it was due to pressure on the brachial plexus between the clavicle and one of the transverse processes: others that it was caused by pressure or squeezing between the clavicle and first rib. From personal experiments it is in my mind exceedingly doubtful if these conditions are anatomical possibilities in normally built babies. I should not be willing to admit such a cause without absolute proof. It is, however, possible that in cases of fracture of the clavicle, or other fracture, some pressure on the brachial plexus may be produced which may cause similar or analogous symptoms to those of ordinary obstetric paralysis. Thorburn mentions this class of cases merely to exclude them from consideration under this title.

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[1] Med. Chron., Manchester, 1885-1886, iii, 146

[2] Boston Med. and Surg. Jour., October 19, 1905.

Setting aside the theories of direct pressure on that portion of the brachial plexus capable of producing these forms of paralysis, we find certain other theories advocated to explain the clinical conditions which later have proved inadequate and have been discarded. In 1892, Chauncey R. Burr[3] attempted to explain these cases as forms of anterior poliomyelitis, a theory which at the time had some plausibility and which in the absence of the objective evidence of the nerve lesion might be hard to disprove.

Carter,[4] in 1893, published an excellent article on this subject. In France experiments were made on the rabbit and on the cadaver by Fieux[5] and these with accounts of cases were reported by him in 1897.

The true condition in many if not all of these cases was made evident (1) by conditions found in traumatic cases of Erb's paralysis in the adult, (2) by experiment on the cadaver and on animals. The first case in which the condition existing in a case of obstetric paralysis was seen was that of Eversmann[6] in 1903. In this case there was found a hard, tumor-like thickening, 0.5 to 0.75 cm. long, at the junction of the fifth and sixth cervical nerves, evidently a callus following a primary injury with laceration of the nerves.

It has now, however, been shown by a number of cases that the injury is due to stretching of the fifth and sixth cervical nerves in the neck. These nerves may be simply stretched without loss of continuity or they may be ravelled out, some fibers being torn apart and some still holding, or they may be wholly torn asunder. The second is probably the most common condition. One nerve may be wholly torn and others partially so. Personally I have had occasion to see this condition in one case.

The history of the various theories which have been held in regard to the etiology and pathology of obstetric paralysis has been so thoroughly abstracted and discussed by Stransky[7] (1902) that further consideration of this subject here is not advisable. Anyone specially interested should consult his most excellent and valuable article.

Etiology. The exact causation of obstetric paralysis, the method in which the pathological condition is produced at birth, is still not perfectly clear. We know that at least in the very large majority of cases the nerves are torn or ravelled out, but the exact process by which this is done is not settled. It seems certain that it is due to traction powerfully exerted and favored by a firm resistance or fulcrum against which the pull is made. The injury to the nerves is undoubtedly favored by placing them on the stretch through the position of the head. A traction on the head in the axis of the body is less injurious than when the traction is made obliquely so that the head is inclined to one side while the traction is made. Rotation of the head to the opposite side also stretches the nerves and traction made while the head is rotated, particularly when it is also inclined toward the shoulder opposite the stretched nerves, strongly favors injury. In order that the full force of the pull should be effective against the nerves, it is necessary that there should be firm resistance. In the case of head presentations this may be produced by the fixation of the shoulder or shoulders; in the case of breech presentations by the resistance of the head.

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[3] Boston Med. and Surg. Jour., 1892, cxxvii, 235.

[4] Ibid., 1893, cxxviii, 434.

[5] Ann. de gynec. et d'obstetrique, 1897, xlvii, 53.

[6] Arch. f. Gynäk., 1903, lxviii, 143.

[7] Centralblatt f. d. Grenzgebeite d. Med. u. Chir., 1902.

Other factors enter into consideration also. It is extremely probable that forced lowering of the shoulder girdle acts unfavorably to the resistance of the nerves and favors this injury. The catching or fixation of the tip of the shoulder produces this lowering, and this we know must often occur. In cases of traumatic Erb's paralysis in the adult the causation is frequently a severe blow upon the shoulder, causing its sudden forced descent.

Asphyxia has been considered as an important factor in the production of this condition. When the patient is asphyxiated the muscles become relaxed, their resistance is removed, and the nerves, no longer having their support, are more easily torn.

STATISTICS. In order to determine practically, as far as possible, the mechanism or causation of the injuries causing obstetric paralysis, I have investigated a considerable number of cases from the Department of Nervous Diseases at the Children's Hospital, Boston. A request was sent to the attendant physicians asking for an account of the delivery and labor. Through the kindness of many of these gentlemen, I have received replies which may be summed up as follows:

Statements from Physicians. I have 43 cases in which the report of the attending physician has been obtained. The character of the labor was described as long, tedious, or difficult in 20 cases; in 6 cases it was noted as normal, and in 4 cases as easy; in 2 cases of the latter as "unusually easy." In 13 cases there was no report as to the character of the labor. It was not possible to distinguish between the protracted labors and those which presented difficulties and obstacles and were classed as "difficult." The two conditions do not coincide, but they are apt to occur together; especially is the difficult labor apt to be prolonged.

The delivery is described as difficult in 15 (very difficult in 7), easy in 1, normal in 6, and is not reported in 21 cases. Forceps were used in 28 cases: in 8 high forceps; in 1 it was noted "medium"; in 2 low. Ether was used in 12 cases.

The head presented in 40 cases, the breech in 3. This would seem, in comparison with statistics obtained from the literature, to be an unusually small proportion of breech presentations.

As to the position of the head, in one case it was noted that the occiput presented. O. L. A., or first position, occurred in 18 cases; O. R. A. in 1 case; the occiput anterior was noted in 1 case, the occiput posterior in 2 cases; O. D. P. in 2, and a face presentation in 2. In 13 cases the presentation is not stated.

In 18 cases it was especially noted that the shoulders were held or offered resistance to extraction, and this fact has been considered by the reporters as unusual and of great importance. On the other hand it was expressly recorded in 5 cases that the shoulders were not held. In regard to traction, it was recorded as used on the neck in 2 cases, on the head in 2 cases, and on the shoulder in 1 case. In 5 cases there was pressure in the axilla by the finger or otherwise. In these cases it is fair to suppose that traction is only mentioned when considerable force was used, at least more than the ordinary amount.

It was mentioned in regard to the arms that in 5 cases they were brought down with difficulty. In 1 case the arm was stated to have undergone great pressure between the body and the pubes. In 2 cases there was no traction. The position of the arms was not mentioned except in 2 cases, in neither of which were they above the head.

These are the results which are obtained from the statements of the attending physicians and are our most reliable data.

From the patients themselves we have in many cases obtained good histories. In addition to the cases in which we have physicians' reports we have 135, making a total of 178.

Statements from Patients. Duration of labor, six hours or under, 12 cases; between six and twelve hours, 8; between twelve and twenty-four hours, 17; between twenty-four and forty-eight hours, 5; forty-eight hours or more, 9 cases.

Labor, severe, difficult, 79 (prolonged, 2); mother died at birth, of nephritis, 1; easy, 1; short, 1; not difficult, 1; normal, 4; and dry, 1.

Presentation. Head, 55; breech, 5; feet, 5; transverse, 3; arm, 2.

Forceps (including 3 on the after-coming head), 93; no instruments, 20.

Anesthetics. Ether, 27; chloroform, 3; no ether, 9. Asphyxia, 42; no asphyxia, 19. Shoulders delayed, 19; head large, 1; caught, 1; body delayed in breech presentation, 1. Arms over head, 2; arms caught, 4.

Injuries. Fracture of clavicle, 5; fracture of arm (humerus), 2; dislocation of shoulder, 4.

As to the limb affected the statistics are: Right upper extremity (breech 2), 92; left upper extremity (breech 1), 72; both, 2; not reported, 11; one case omitted as not obstetric paralysis, 1.

It has been suggested that in head presentations the paralyzed arm corresponded to the shoulder which was held under the pubes. This shoulder being fixed, the strain came upon the nerves on this side and the corresponding limb was injured. Hence in the O. L. A. position in which the right shoulder is held under the pubes the right upper extremity should be the one paralyzed. This theory is not confirmed by my observations, as out of the 17 cases of O. L. A., in 8 the right upper extremity was the one involved, and in 9 it was the left upper extremity.

CLINICAL CONDITIONS. The clinical conditions vary in different cases of obstetric paralysis, according as we have the upper-arm type (Figs. 1, 2 and 3), the lower-arm type, or the type which involves the whole extremity. I shall consider in this paper only the upper-arm type. In the larger number of cases of this character we find involved the deltoid, biceps, brachialis anticus, the supinator longus and perhaps the brevis, the infraspinatus and supraspinatus, and serratus magnus. Occasionally there are other muscles involved, as the rhomboids, the subscapular, and probably the coraco-brachialis and subclavius.

If the other roots have been wholly or partially involved there is more or less affection of other muscles of the limb, involving the movements of the arm, forearm, or hand, and the case approaches the third type. The clinical results of involvement of these muscles can be more or less logically anticipated.

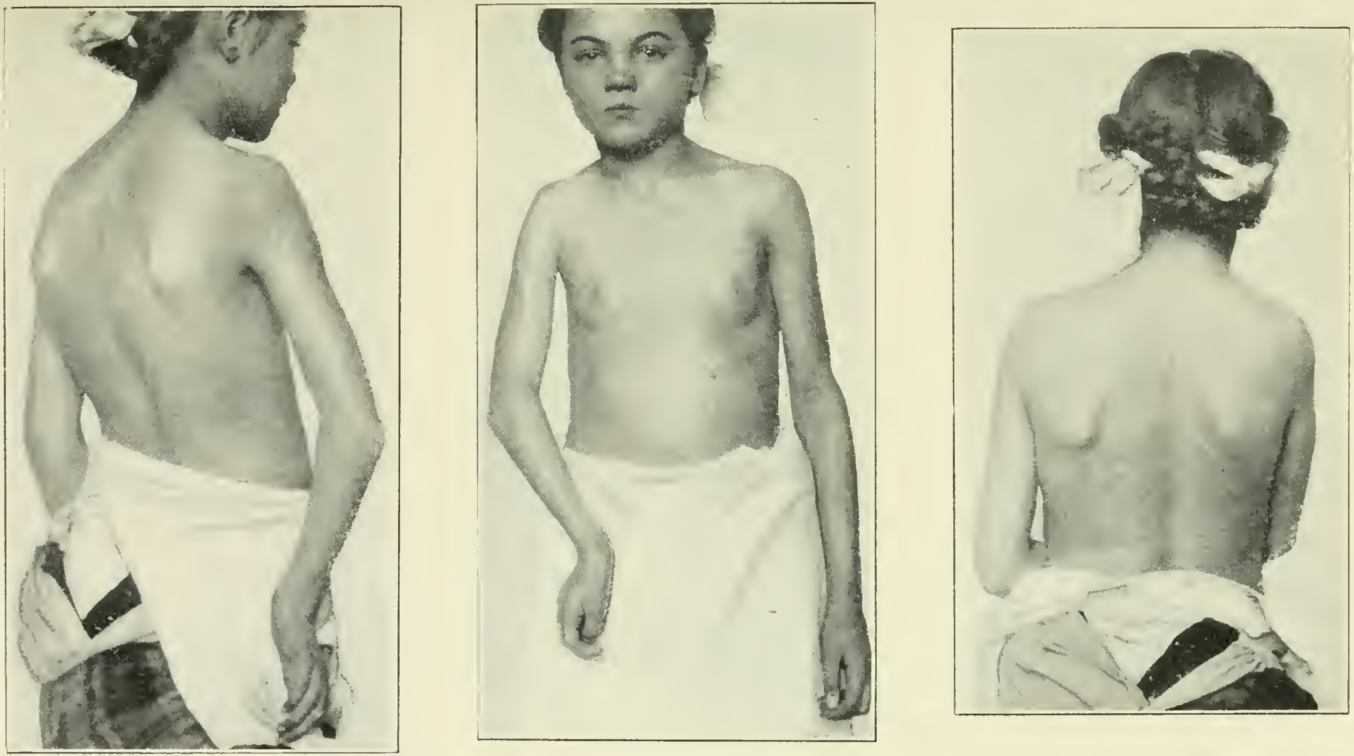
Clinically we find the following conditions:

In the early stages the upper extremity hangs limp, extended by the side, and the whole limb is internally rotated. In typical cases the grasp is unimpaired in this type, and the wrist is not affected. The forearm cannot be supinated beyond the median line. Flexion of the forearm on the arm is weak and poor, not wholly absent, extension not affected. The arm cannot be elevated at the shoulder nor can it be rotated outward; inward rotation is impaired, inasmuch as the limb is held more or less fixed in the position of internal rotation. Passive movements are now perfectly free.

The anatomical causes of the typical position of the upper extremity and of its inability to perform certain movements in obstetric paralysis of the upper-arm type are very interesting.

Inasmuch as the extremity assumes at once on the outset of the paralysis its typical position, it is plain that this must be due to the uncontrolled action of the unparalyzed muscles. The three principal elements of the typical position are the internal rotation of the arm and forearm, the extension of the forearm on the arm, and the hanging of the arm due to inability to raise it at the shoulder.

(*a*) The internal rotation of the arm is due in most cases to the action of the pectoralis major and the subscapularis. Perhaps it is more accurate to say that the pectoralis major adducts the arm and brings it forward on to the breast while the subscapular rotates



Figs. 1, 2 and 3.—Obstetric paralysis, upper arm type; characteristic position of the arm it. The latissimus dorsi and teres major assist in the internal rotation of the arm.

(*b*) The pronation of the forearm and hand is caused partially by the internal rotation of the arm. It is also caused undoubtedly by paralysis of the supinator brevis, and especially by the paralysis of the biceps.

(*c*) The extension of the forearm on the arm is due to the paralysis or weakness of the flexors of the forearm in the arm, the biceps, brachialis anticus, and coracobrachialis.

(*d*) The hanging of the upper extremity from the shoulder is due chiefly to the paralysis of the deltoid. The weight of the limb itself causes this when its support is removed.

If we examine more closely into the involvement of the muscles in the upper-arm type of obstetric paralysis we shall be in a better position to understand, at least in part, the conditions which are found.

We shall consider the muscles in turn. (The nerve supply is given in parentheses after each muscle.)

I. *Serratus Magnus.* This is usually involved. It has been stated by some writers that it is never involved, but this I believe to be a mistake. (Posterior thoracic nerve.) Angel-winged paralysis.

II. *Subscapularis.* It is practically impossible clinically to state in most cases whether this muscle is involved or not. In many cases it seems to be normal. (Subscapular.) Inasmuch as the internal rotation of the arm is presumably in most cases caused by this muscle, the clinical evidence is in favor of its not being weakened or paralyzed.

III. *Supraspinatus* and

IV. *Infraspinatus.* These muscles are usually involved and often markedly atrophied. In the typical cases they are involved and atrophied. (Suprascapular.)

V. *Teres Minor* and

VI. *Teres Major.* The teres minor is quite certainly usually involved, although this is not always apparent to the eye. (Circumflex.) There is more doubt in regard to the teres major, but it is probably usually affected. (Lower subscapular.)

VII. *Deltoid,*

VIII. *Biceps,*

IX. *Brachialis Anticus.* These three muscles are always involved. The deltoid is the most affected, as a rule, of any muscle, and is in all typical cases atrophied. The biceps and brachialis anticus are always weakened and are atrophied in all severe cases.

X. It is often said that the *coracobrachialis* is not involved. This is somewhat difficult to prove. (Musculocutaneous.)

XI. *Triceps* not involved. (Musculospiral.)

XII. and XIII. *Supinator longus* is involved in all typical cases —usually much involved. (Musculospiral.) The *supinator brevis* is said to be involved in many cases. (Posterior interosseous.)

XIV. *Subclavius.* It has been said that the subclavius is sometimes involved. (Cord of fifth and sixth.)

XV. *Pectoralis Major.* In large part not involved. In some cases the upper portion is distinctly atrophied. (Anterior thoracic.)

XVI. *Pectoralis Minor.* No clinical evidence of involvement. (Anterior thoracic.)

SUMMARY. Typically involved are the deltoid, biceps, brachialis anticus, supinator longus, supraspinatus and infraspinatus, serratus magnus. Probably usually or frequently involved in addition are the teres minor, coracobrachialis, and supinator brevis. The subclavius is probably sometimes involved. The upper portion of the pectoralis major is sometimes involved. The subscapular is probably not often affected. The trapezius is apparently often weakened.

The cause of the inability to perform certain movements in this affection is the paralysis of the muscles by which these movements are performed. It is a task of considerable difficulty to determine what muscles are involved in each case.

The *general rules* are as follows: The inability to raise the arm to the level of the shoulder is due in large part to the paralysis of the deltoid and the supraspinatus. The serratus magnus and trapezius aid in performing this movement in the healthy person.

The inability to elevate the arm from the level of the shoulder to the full height is due chiefly to paralysis of the serratus magnus. In the healthy person the deltoid and trapezius assist in this movement.

The inability to rotate the arm externally is due to the paralysis of the infraspinatus and teres minor. The posterior portion of the deltoid helps in the healthy to perform this movement.

The inability to rotate the arm internally is due to the fact that the internal rotators are already fully contracted.

The weakness and inability to flex the forearm strongly is due to the weakness or paralysis of the biceps, brachialis anticus, coracobrachialis, and supinator longus.

The inability to supinate the forearm is due in part to the position of the arm and in part to weakness of the biceps and the supinator brevis.

The inability to extend the forearm fully on the arm which is present in many old cases is due sometimes to actual atrophy and malformation of the bones, and at others apparently to a contraction of the capsule of the joint, especially the anterior portion.

As the paralysis continues new conditions arise. On examination of the older cases we ordinarily find an atrophy of the scapular muscles well marked, sometimes a rotation of the scapula or a standing off of its internal border from the ribs, which might be caused by weakness of the trapezius or of the serratus magnus. There is usually marked atrophy of the deltoid and some atrophy of the whole extremity, most apparent in the arm, somewhat in the forearm, while the intrinsic muscles of the hand are little or not at all affected.

The atrophy of the limb is, in the older cases, not confined solely to the muscles, but the bones themselves are affected and the whole limb may thus be smaller than the corresponding one. We may find at the shoulder a loose joint, though this is less extreme than in some cases of anterior poliomyelitis. I would here call attention to the fact that the atrophy and paralysis of the arm muscles is less complete, as a rule, than in the old cases of anterior poliomyelitis. This may be due to the fact that in the laceration and ravelling of the nerve cords some fibers are left unhurt or are not totally destroyed.

In these cases other conditions develop or appear. It is common to find evidences of adhesions between the scapula and the structures of or around the shoulder-joint, so that in partial passive elevation of the arm the scapula is moved, and as the arm becomes elevated above the level of the shoulder the scapula is drawn into the axilla. Inability to produce absolute extension of the forearm occurs in many cases. Its cause will be discussed later.

Passive supination of the forearm is sometimes impossible, apparently on account of adhesions.

ATROPHY AND MALFORMATION OF BONES. In addition to the muscular atrophy we find evidence of atrophy or non-development of the bones. The limb may be actually shortened in comparison with the healthy one.

Shoulder. Displacements of the head of the humerus are not rare. The head of the humerus is usually displaced anteriorly. There is an anterior subluxation. It may, however, be displaced posteriorly —posterior subluxation.

These displacements are due apparently to the contractions of the unparalyzed muscles and possibly of some of the tissues surrounding the joint. There is no evidence of any contracture of the paralyzed muscles.

The paralysis is always flaccid. The capsular ligament surrounding the joint is undoubtedly stretched.

I had long supposed the anterior subluxation to be due chiefly to the contraction of the pectoralis major, the upper portion of which can be felt hard and tense, holding the arm fixed in internal rotation. I am inclined to believe now, however, that the subscapular plays an important part in this connection.

The cause of the posterior subluxation is more doubtful. The paralysis of the supraspinatus is undoubtedly the most important factor. The latissimus dorsi and the sternocostal portion of the pectoralis major by their contraction tend to separate the head of the

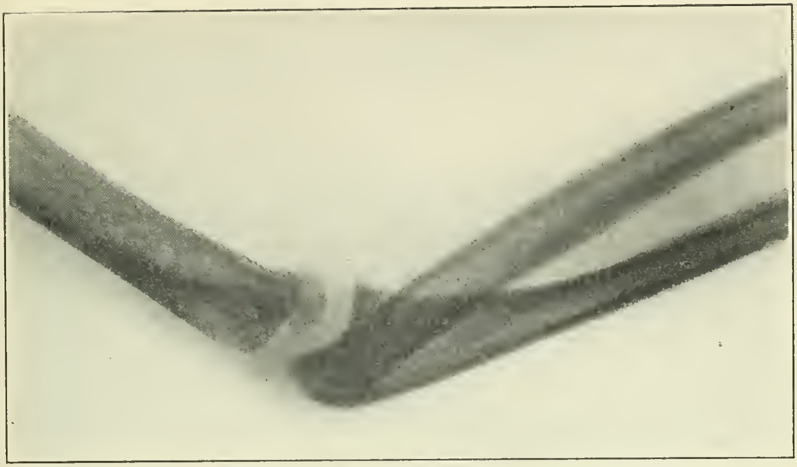


Fig. 4—Elbow-joint, showing backward dislocation of the radius.

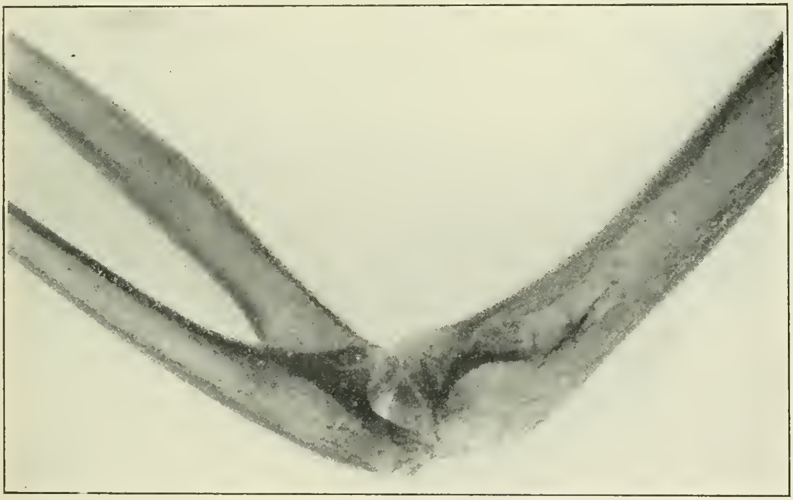


Fig. 5.—Elbow-joint, showing lack of development of the olecranon and impairment of extension by bony contact.

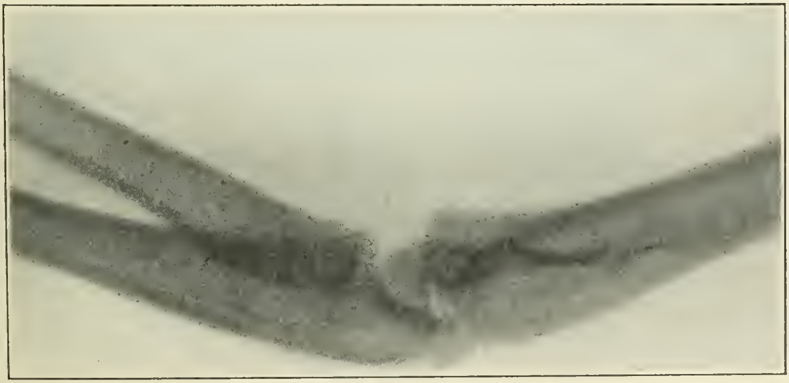


Fig. 6.—Lateral displacement of the elbow-joint.

humerus from its socket, and if unopposed may cause subluxation. They are opposed by the coracobrachialis and the long head of the triceps.

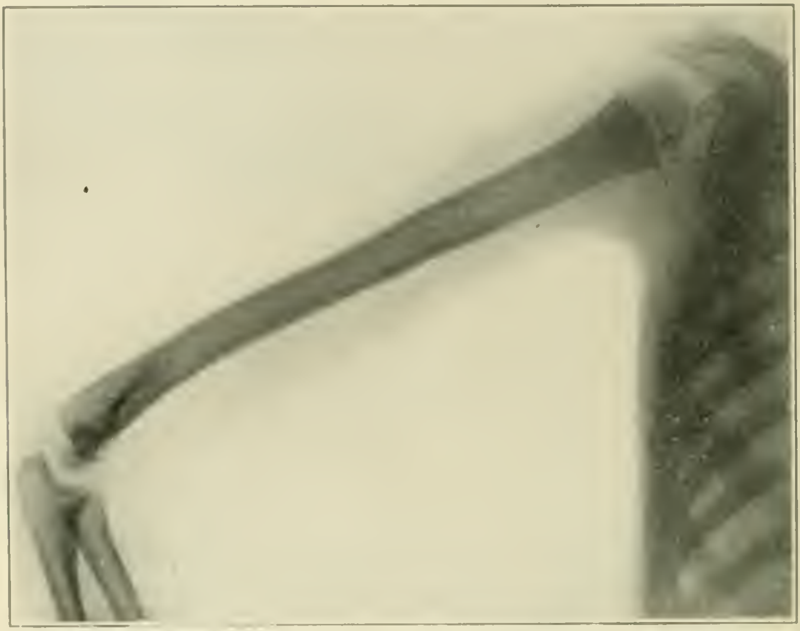


Fig. 7.—Posterior subluxation of the head of the humerus, with marked atrophy of the bones of the arm and forearm.

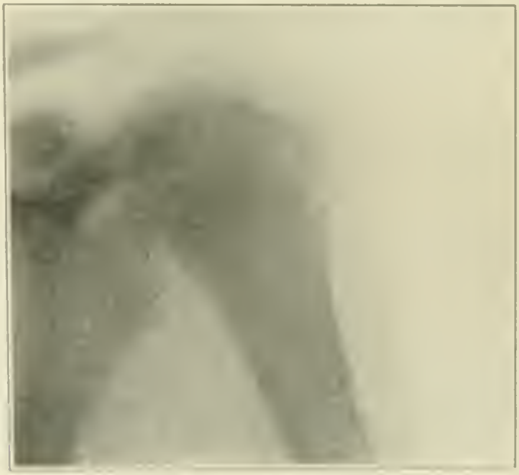


Fig. 8.—Anterior subluxation of the humerus.

Stone (1900) states that backward dislocation of the humerus is due to the stretching of the posterior portion of the capsule, which is weaker than the anterior portion, and that this stretching is the result of the strong internal rotation of the humerus, aided by the backward and downward pull of the latissimus dorsi.

Elbow. It is usually the case that extension of the forearm on the arm is impaired. Full extension, either active or passive, is not possible, resistance being encountered toward the end of extension.

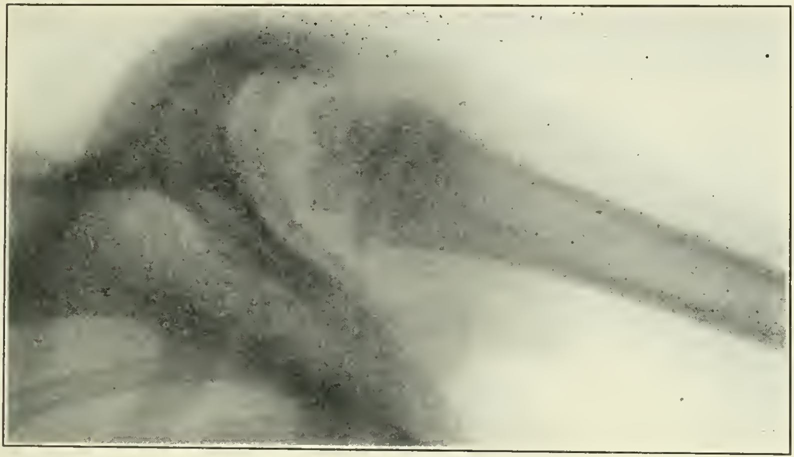


Fig. 9.—Posterior subluxation and atrophy of the head of the humerus.

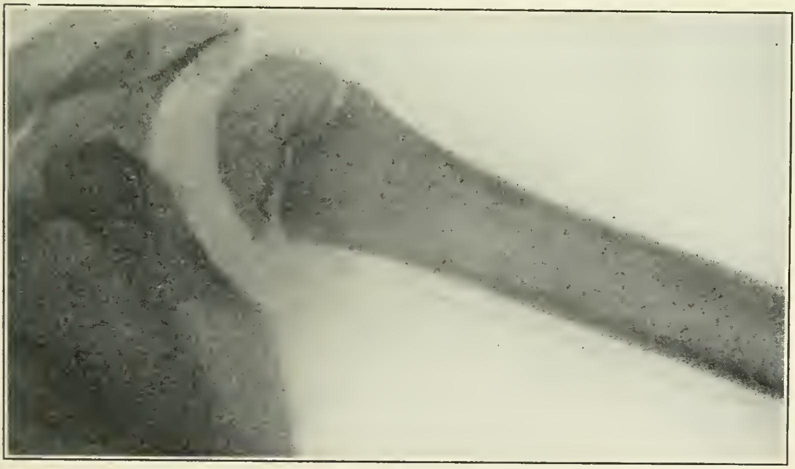


Fig. 10.—Posterior subluxation of the humerus and dropping of the humerus.

The forearm cannot be extended fully in the axis of the arm, but remains at an obtuse angle of 170 to 175 degrees.

This may be often due to the contraction of ligaments or of the tissues surrounding the joints, but it is at times due to atrophy and malformation of the bones themselves, as may be seen in the accompanying *x*-ray prints.

In Fig. 4 there is a backward dislocation of the head of the radius, and as a result of this the head is much elongated and thinned. The impairment of motion occurs less sharply than in Fig. 5.

Fig. 5. The limitation here is due to the lack of development of the olecranon, which does not extend backward and upward to the normal amount. The result is that when the elbow is extended the tip of the olecranon impinges on the articular surface of the humerus instead of sliding up behind its lower end, and thus by actual bony contact prevents full extension. In this case the actual impairment of movement appears increased to the observer on account of the curving forward of the lower end of the shaft of the humerus, which in addition to the real impairment produces an apparent flexion.

Fig. 6 is another excellent radiogram of an elbow in this affection. There is some lateral dislocation and the bones hitch on extension.

Fig. 7 is a radiogram showing both the shoulder-joint and the elbow-joint. There is marked atrophy of the bones of the upper extremity, and in this case there was a posterior subluxation of the head of the humerus.

Fig. 8 shows anterior subluxation of the head of the humerus, which is about the normal size and is to be compared with

Fig. 9, which shows marked atrophy of the head of the humerus in a case with posterior subluxation, and retarded development of the epiphysis.

Fig. 10 is a radiogram of another case of posterior subluxation, showing the dropping of the head of the humerus from the scapula on account of paralysis of the deltoid and stretching of the capsule from the weight of the limb. This is from the same patient whose elbow is shown in Fig. 5.

TACTILE AMNESIA.

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Not infrequently patients suffering from hemiplegia are unable to recognize by touch objects in the paralyzed hand. This must occur if there is complete insensibility to all forms of sensory stimuli. But it also occurs when there is only partial insensibility, when only certain kinds of sensations are unperceived. Mere tactile anesthesia—that is, the inability to feel contact on the skin without pressure or at least with no more pressure than is made by a wisp of cotton—does not prevent the recognition of objects. When the anesthesia affects the underlying tissues so that deep pressure is not felt, the patient not only does not know what he has in his hand, but does not know that he is grasping anything. More important, then, than light touch in the recognition of objects is the ability to know the position of the fingers, to differentiate weight, to locate the point touched, and to recognize the number of points of contact. Failure in these regards, even if mere touch sensibility is preserved, leads to true astereognosis. In healthy men objects are distinguished much more accurately by handling them than by contact with any other part of the body. Some objects (apart from recognition by taste) can be recognized by contact with the lips, tongue, and mucous membrane of the cheeks. The ability to recognize objects in contact with the feet is almost *nil.* This is probably largely owing to the fact that throughout life no attention is given to educating the sensibility of the feet and partly to the lack of prehensile power in the toes. Armless men who have learned to use their feet more or less well as their hands have a greatly developed power of recognition of objects by them. Persons who have been hemiplegic from birth or early childhood are sometimes astereognostic in the palsied arm from lack of training.

These few preliminary words about astereognosis are necessary because it is often confused with tactile amnesia. In the latter sensibility to all forms of stimuli is preserved, but the patient, though knowing he has something in his hand, yet cannot recognize what it is. It is not that he cannot, though knowing what the object is, recall its name (tactile aphasia), but he does not know its nature or uses, knows nothing about it. Of course, the recognition of an object is a psychical process. A great number of sensations from the skin and underlying structures, muscles, and joints are combined and form the perception of a key, an apple, or whatever the object may be. Though the judgment is psychical there must be stored, somewhere in the cortex, memories of passed tactile sensations of other keys, apples, etc. Disease destroying this part of the cortex or cutting it off would lead to tactile amnesia. The location of this centre is yet not clearly and definitely known, but it probably is in either the posterior part of the parietal lobe or the anterior part of the convex surface of the occipital lobe. The frequency of the association of the symptom with word blindness and mind blindness makes it probable that the centre is near the higher visual centre. It cannot be identical with the centre for common sensation, because tactile sensibility is not affected. The frequent occurrence of the symptom with mixed sensory aphasia due to a unilateral lesion would indicate that the centre is unilateral. It must be posterior to the fissure of Rolando. The symptom never occurs if the area of disease involves only the prefrontal lobe. I have never seen it in pure motor aphasia, in uncomplicated left-sided hemiplegia, nor in right-sided hemiplegia unless there was also a greater or less degree of word blindness or object blindness or some type of sensory aphasia. It is frequent in pure mind blindness and in mind blindness occurring as an early symptom of dementia caused by diffuse organic brain disease. There may, however, be complete mind blindness, an absolute inability to recognize any object by sight, without any tactile amnesia.

In differentiating it from astereognosis it must be remembered that there is no disturbance of primary sensibility and that both sides are affected. I have never seen it as an entirely isolated symptom and do not know whether it occurs. It is sometimes an early or late symptom of organic dementia. Its presence or absence cannot of course be determined in severe cases of total or of mixed sensory aphasia, because it is impossible to make such patients understand what they are to do when an object is put in the hand. The only patients suitable for examination are those in whom the aphasia is partial or in whom recovery has gone far enough for the patients to understand short and simple commands and to use language enough to show whether they know what they are grasping. Nothing can be judged from the patient's general conduct whether the symptom is present or not, because vision helps him in all he does. It can only be discovered by examination.

CASE I.—*Complete tactile amnesia associated with sensory aphasia, hemiplegia, word blindness, and partial object blindness.*

J. M., seventy years old, was admitted to the hospital in December, 1902, with right hemiplegia and jargon speech. His previous history could not be obtained, but he must have been ill quite a long time, because the paralyzed arm was contractured and his gait was that of an old hemiplegic. Spontaneous speech, that is, speech as a method of expressing ideas arising within his mind and not merely words recalled reflexly in response to external stimuli, was an unintelligible jargon. There was not complete word deafness. He understood and obeyed short and simple verbal commands such as "stand up," "sit down," "walk," "stand still," but failed to obey any order in the least complicated. His difficulty seemed to be not so much in understanding single words as in carrying the first words of a long sentence in consciousness until the last were spoken. Words made only a momentary impression on him. He understood gestures and pantomime fairly well. By the combined use of spoken words, gesture, and pantomime I was able to make him understand he was to tell me the names of objects shown him and put in his hands. He named at sight a box, spoon, fifty-cent piece, ten-cent piece, watch, book, penholder, candle, and scissors. He could not name and gave no evidence of recognizing a fork, cigar, key, or knife. He failed to recognize any object put in either hand. He did not understand written or printed words and could not even write his name. His condition remained unchanged for two months, when he died of chronic Bright's disease.

CASE II.—*Transitory hemiplegia and permanent jargon aphasia with partial tactile amnesia.*

J. C., seventy-six years old, was admitted to the hospital in January, 1906. Two and one-half years before he had had a stroke, causing transitory difficulty in speech. The day before admission he fell unconscious and so remained a few minutes. When consciousness returned speech was so mixed that he could not be understood and he was palsied on the right side On admission speech was so mixed that it was impossible to understand him. He was not entirely word deaf because he obeyed simple verbal commands, but he failed to obey any complicated order. There was no hemiplegia. Three days later he had improved a great deal. He wrote his own name, but nothing else. (He had been taught to read and write.) He looked blankly at written and printed words and made no attempt to obey written orders. He named at sight most numbers below 100, but failed on all larger ones. He recognized and named all the letters except U. He copied a few short words, but soon became confused and ceased to make any further effort. He did not understand the meaning of the words he copied. Spontaneous speech had improved very much. He could carry on a simple conversation for a few minutes, but soon became mentally tired and confused and then his speech became very mixed. He recognized and named at sight almost all the common objects shown him. Some objects put in either hand he recognized and named; some he recognized, as was proved by his description of them, but could not name (tactile aphasia); others he did not recognize. Some objects recognized by touch were not recognized by sight, and vice versa. There was no anesthesia of any kind in either hand. When he was discharged several weeks later spontaneous speech was quite good, but he still became confused if he talked for a long time. His disability in recognizing objects by touch and his inability to read and write remained unchanged.

CASE III is very complicated because in the paralyzed hand there was sufficient sensory disturbance to produce astereognosis, while in the left, with normal sensibility, the patient was able to recognize and name some objects but not others—partial tactile amnesia.

C. D., forty-five years old, was admitted to the hospital in April, 1905. Two months before admission he suddenly became confused, mixed in speech, and, in the course of a few hours, lost power in the right arm and leg. He had a right-sided hemiplegia. He could walk, but dragged the right leg, and the right arm was contractured. There was slight palsy of the lower right face. He had marked disturbance of spontaneous speech. On beginning to talk he did well, but soon tired, became confused, and then speech was unintelligible. He understood and obeyed simple verbal commands and answered simple questions by simple words. He became much mixed on attempting to use long sentences in talking. He recognized and named most objects at sight; a few he described by a paraphrase or a not strictly accurate name, that is, a teaspoon was a "thing to eat with" and a cork was a "stopper." He recognized by sight all the letters and could read aloud and understand the meaning of short words. He could copy and sometimes understand words he copied. He recognized numbers of not more than two figures. He could write his signature C. B. Blank, but not his first name, Charles. He could write most letters at dictation, but no words. He could not recognize any object put in the right hand. Though the right arm was palsied, there was enough power in it to grasp and "actively" feel objects. Sensibility to touch was preserved, but though he located a single point of contact fairly well on the right hand he could not distinguish whether he was touched in one, two, or three places simultaneously unless they were very wide apart. When touched in several places at once he usually located one fairly well, but apparently had no knowledge of the others. He knew in a way, but not accurately, the position in which the fingers of the right hand were passively put, because he could put the fingers of the left hand in about the same attitude. On the left hand there was no disturbance of sensibility of any kind. In it he recognized a few objects, but failed entirely to recognize many.

CASE IV.—*Slight hemiplegia, mixed sensory aphasia, partial object blindness, and partial tactile amnesia.*

S. B., fifty years old, was admitted to the hospital in April, 1904. He had an old right-sided hemiplegia involving the face. He could walk a little and move the right arm slightly, but not use it for any purpose. On admission his speech was such a jargon that it could not be understood, and he obeyed no verbal commands. By the third day he had improved enough to understand and obey simple commands. He understood that he was to tell me what the things were I showed him and put in his hands. At sight he recognized and named a watch, handkerchief, one-cent and five-cent pieces, two and five-dollar bills (as money, but not the denomination), a spoon, a cigar, bunch of keys, and a cup. Many other common objects he did not recognize. By touch in the right hand he recognized and named a penknife, key, and watch, but nothing else. In the left hand he recognized nothing. If he handled and looked at an object he did not recognize and at the same time was told its name he did not recognize the name. Speech improved a great deal, but his visual and tactile disturbance continued until death, which occurred some weeks later from uremia.

CASE V.—*Transient right hemiplegia, aphasia, and tactile amnesia.*

W. C., sixty years old, was admitted to the hospital in August, 1904. On January 14, 1904, he awoke paralyzed on the right side and with inarticulate speech. He improved, but the day before admission speech again became affected. He had never been taught to read and write. The lower right face was slightly palsied. At the beginning of the examination he answered questions without difficulty, but soon tired mentally and became confused and misused words. There was no disturbance of sensation. He recognized and named almost all objects shown him, but could not recognize or name any object put in either hand. In a few weeks he could talk fairly well and recognized and named all objects by touch. He died some months later without any return of palsy or aphasia.

CASE VI.—*Marked dementia, mind blindness, slight amnesic aphasia, and partial tactile amnesia.*

J. M. was an educated man, fifty-seven years old, and had been actively engaged for years in literary pursuits. He had been a heavy drinker, but so far as known had never had syphilis. Early in 1898 he began to be irritable and easily angered and careless about his business affairs, but nothing was suspected of any serious illness until July, when he was suddenly taken ill, becoming delirious and vomiting frequently. He continued to be very excited for several days and then became quiet, but his speech was "a mere gibberish." He was not paralyzed and his speech slowly returned to normal. A similar attack recurred in September. He was alleged to have been blind after both attacks for a short time, but it probably was mind blindness, for he avoided obstacles in walking. He never worked after the first attack, lost all ambition, and was easily controlled.

His general nutrition was good. Gait and station were normal. There was no paralysis of the extremities or cranial nerves. Both knee-jerks were spastic. His voluntary speech was somewhat affected. For the most part he spoke well, but occasionally he would fail entirely to recall a word, or would misuse a word or be compelled to make a paraphrase. He recognized his errors immediately. He understood all that was said to him, but after conversing for some minutes would become a little confused and then his speech would become much worse. He could repeat any word said to him. He could write a few words or short sentences from dictation equally well with the eyes open or shut, but could not read what he had written. Writing soon fatigued him and then he failed entirely. He could not read any writing or print. He could not recognize any object by sight, but knew there was something before him and whether it was longer than broad, or whether it was larger or smaller than some other object shown him at the same time. He recognized the nature and uses of a few objects on handling them, and described them by paraphrase, but could not recall the names. The great majority of objects he did not recognize by touch at all. There was no disturbance of sensibility in either hand. Dementia at first slight, later became quite marked. He had then no realization of the fact that he was a seriously ill man. On the contrary he was overhappy, but had no definite delusions of grandeur. He had lost all intellectual interests. There was a double murmur at the aortic cartilage, hypertrophy of the heart, and the radial and temporal arteries were much thickened. He had no subjective symptoms of heart disease. The most accurate diagnosis of his case is organic dementia caused by diffuse cerebral degeneration. Though the mind blindness, aphasia, and tactile amnesia increased his dementia, they were not its primary cause, because it began before they appeared.

CASE VII.—*Word blindness and partial tactile amnesia coming on after a sudden hemiplegia and followed by complete dementia, caused by arterial sclerosis and brain tumor.*

The patient was a merchant, fifty-five years old, who about nine months before I first saw him suddenly became hemiplegic. He was a fairly well-built, elderly man. His gait was slightly paralytic and at the same time spastic. There was not marked hemiplegia, but slight athetoid movements were constant in the right arm and were made worse by any voluntary muscular effort. Both knee-jerks were increased, the right being the greater. The right grip was weak, the left good. Babinski jerk was present in the right foot. Speech was normal and he understood everything said to him. He could not understand writing or print except single letters and a few short words. He recognized and named all but a very few objects at sight. He could recall the names of only a very few, though he recognized them. He could recognize and name a few objects by touch; others he recognized, but could not name; but by far the greater number he neither recognized nor named. He could not write with the right hand and any attempt to do so greatly increased the athetoid movements. With the left hand he could write many letters and a few short words. In the left hand sensibility was normal, but in the right two points of contact had to be widely separated to be distinguished. The right pupil did not react to light, the left reacted feebly; both reacted well with convergence and accommodation. There was quite marked optic atrophy on both sides.

A few months later he had a general epileptiform convulsion, and several weeks later awoke one morning speechless except for the words "yes," "no," and "doctor." In the course of a few days speech returned. Several months later he had six general convulsions in one day. Once he had an attack of transitory blindness and jargon speech. As time passed he grew weaker on the right side and his mental state deteriorated. His memory became very poor. He failed to recognize relations and friends, sank into complete dementia, and died.

At necropsy a large tumor was found infiltrating the whole left parietal lobe and the anterior part of the occipital lobe. It did not pass the fissure of Rolando anteriorly. Microscopically it proved to be a sarcoma. The cerebral arteries were all very atheromatous, and many small areas of softening were present in both hemispheres. There was mitral and aortic valvular disease, hypertrophy of the heart, and chronic interstitial nephritis. The lesions were so widespread that the case could not be used to locate any function.

DIFFICULTIES IN THE DIAGNOSIS OF BRAIN ABSCESS.

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Spiller, in a very able article, says that the "diagnosis of brain abscess depends chiefly upon the signs of some more or less rapidly developing lesion of the brain, with the discovery of a purulent process in the body, or a wound of the head. Abscess of the brain without any evidence of pus elsewhere may be extremely difficult to diagnose." Aurists are rarely concerned with cases other than those having a history of suppuration with the principal focus in the ear. It is not the purpose of this paper to take up the diagnosis of brain abscess, but rather to point out some of the difficulties. Almost every operation is undertaken with some doubt in the mind of the operator. Nothing is clear until the abscess cavity is opened or an autopsy shows the lesion.

The cardinal symptoms of cerebral abscess are neuroretinitis or choked disk, subnormal temperature, headache, chill, vomiting, slow pulse, slow cerebration, stupor, high leukocytosis with high polymorphonuclear percentage, and cerebrospinal fluid free from pus. Cerebellar abscess frequently gives no characteristic symptoms. The diagnosis is best given in Dana's cerebellar seizure, viz., sudden unexpected attacks of extreme vertigo, roaring in the head, relaxation of the limbs, and falling to the ground in a semiconscious state. Sachs calls attention to a symptom that may help at times, that is, the early involvement of the facial nerve, not paralysis, but reactions of degeneration. He claims that the early development of reactions of degeneration in the facial nerve indicates the probability of an abscess in one of the lateral lobes of the cerebellum rather than a sphenoidal abscess.

Unfortunately our cases usually do not present groups of symptoms sharply defined. The changes in the fundus of the eye are generally late; their absence, therefore, is not a help. The temperature is frequently subnormal, but sometimes does not fall below 101° F. The slow pulse is a reliable guide when present, but in some cases it never goes below 80, keeping in perfect accord with the temperature. The headache and slow cerebration are nearly always present, with stupor as a later symptom. Kernig's sign may be present, but is more generally absent; it is almost constant in meningitis.

It may be said that localizing symptoms of brain abscess are practically never present if the abscess be small, and they are frequently absent even when the abscess is of considerable size. Abscess of the frontal lobe is usually attended with no localizing symptoms, unless it is situated about the base of the third frontal or ascending frontal convolution. If the abscess is large the pupil of the same side is usually dilated; if small, the pupil may be sluggish and contracted

If a temporosphenoidal abscess be small there are usually no localizing symptoms; when large the adjacent centres and nerves are likely to be affected, either by pressure or inflammatory action. Hemiplegia on the side opposite the lesion is occasionally found in large abscess. Facial palsy may be caused by lesions in the tympanic antrum, or it may be due to a cortical lesion. In the former case the paralysis is complete; in the latter, the patient is usually able to move the eyelid. If due to antral lesion it is on the same side; if due to cortex involvement it is on the side opposite the lesion.

Aphasia has been noted in abscess of the left temporosphenoidal lobe; its presence is a help in diagnosis, its absence has no significance, as was noted in 3 of my cases. If it be sensory aphasia, word deafness, the lesion is probably in the posterior part of the lobe; motor aphasia, in the anterior portion. Paralysis of the third nerve is sometimes seen in large abscess of the temporosphenoidal lobe, and when complete the picture is characteristic: ptosis, external strabismus, and fixed and dilated pupil. The eyeball can be moved only outward and slightly downward. The presence of any of these symptoms is a help in diagnosis and localizing of the abscess; their absence has no clinical significance.

It is seen, therefore, that no symptom is pathognomonic; that diagnosis is nearly always difficult, and must be made by the general clinical picture. The patient almost always has an unhealthy, septic look, but this, too, may be absent. The bacteriological findings are helpful only indirectly, as up to the present time no definite conclusion can be drawn.

When meningitis and brain abscess are found together the diagnosis may be difficult or impossible. Such a case came under my care. The possibility of abscess was suspected; at the time of operation the bone over the dura was found to be destroyed for an area about 2 x 3 cm., but the dura was covered by healthy granulations. These were cleared away and the membrane was found to be considerably congested, but there was no opening, no marked pressure, and there was a good pulsation. Following operation the patient showed a typical clinical picture of meningitis. The temperature ranged from 102° to 103° F.; the pulse from 100 to 110 and was steady. Kernig's sign was present. Lumbar puncture gave apparent relief and showed some pus with streptococci. There was no change in either eye, and no stupor; but the patient was irritable and rather excitable. There was no vomiting; no chill; no aphasia. The patient died seven days after the operation, and the autopsy showed a small abscess in the left temporosphenoidal lobe.

In another case a laborer had had a history of suppuration of the middle ear dating back twenty years. For about a week before admission to the hospital he had complained of intense headache. On the day of admission he suddenly became unconscious just after eating his breakfast. He was brought in threshing his arms about like flails and had to be strapped. He talked in a rambling manner. Once, after admission, his pulse was 62. There were no changes in the eyes; he vomited once. In the absence of all other indications the brain was explored. There was very great pressure and full pulsation; but after a careful search I failed to locate the abscess, and as the patient apparently was sinking rapidly he was sent back to bed. He died ten hours after the operation, and the autopsy showed a small abscess below the first temporal convolution on the left side.

Another patient was seen two hours after admission to the hospital. Her mental state was such that a reliable history could not be taken. She was constantly crying out, "Oh, my head! Oh, my head!" When spoken to sharply she would rouse and answer questions sometimes apparently consciously, at other times in a rambling fashion. She referred her pain to the left side.

Apparently she had had a discharge from both ears for some years. Once after admission, with a temperature of 101.6° F., her pulse dropped to 64. There was no chill and no vomiting. The eye-grounds showed both disks well defined, but the left not quite so sharply. The disk and retina were transparent; there was no change in the vessels. They had the appearance, in the opinion of Dr. Wm. T. Shoemaker, of the probable initial stage of meningitis.

A radical operation was performed at once, but no communication with the cerebral cavity was found. It was thought best to study the patient before going farther, since all of us have seen cases with symptoms of abscess fully as pronounced, or even more so, clear up under the radical operation. Just a short time previously I had operated on such a patient whose condition cleared up nicely, even with a pulse of 60, marked changes in the eye-grounds, sluggishness and vertigo, vomiting and headache.

The day following the operation the patient was much improved in every respect. She was bright and intelligent and hungry and each day showed apparent improvement. She now gave the following history: Scarlet fever at five years, with diminished hearing following. Diphtheria at twelve years, with discharge at irregular intervals from both ears. Had had a paracentesis performed seven years ago. One week before admission she had a throbbing frontal headache; vomited a few times and was very restless. Says there was considerable swelling over the mastoid, but there was none when admitted. The differential blood count was rather startling. Leukocytosis of 15,000 with polymorphonuclear percentage of 89. Cultures showed a short chain streptococcus. This with her variable pulse was sufficient to call for careful watching. But she felt so well and looked so well that my confidence in the polynuclear percentage was wavering.

On the seventh day she complained of headache again; was not feeling so well; when I saw her in the early afternoon she looked septic; her pulse had touched 52, and she was distinctly worse. She was at once taken to the operating-room and an abscess of the temporosphenoidal lobe, holding 75 c.c. of pus was evacuated. Her recovery from this time on was uneventful.

This patient was right-handed, but there was no aphasia. In the 3 cases mentioned there was complete failure of a possible localizing sign, aphasia, for in a right-handed person we would usually find the centre of speech on the left side. The second case was a boy admitted to the Children's Hospital in the service of Dr. Randall. It was owing to his absence from town that the patient came under my care, and it is due to his kindness that I am permitted to report it.

The patient was admitted on the afternoon of July 9, and operated on the following day. The radical operation was done on the right side. Sinus involvement was suspected and a careful search was made, but the parts appeared to be in good condition. The boy did very well until the afternoon of the 14th, when, without chill or other sign, the temperature dropped to 98°, and then rose rapidly to 105°. Owing to the fact of the sinus having been manipulated the possibility of a simple phlebitis was considered and it was decided to wait. But during the evening and early morning hours there was a chill and the temperature dropped to 97° and rose to 106.8° F. He was operated on at noon, and a thrombus was found in the jugular bulb with pus in the sinus. During the first four or five days following operation he appeared to improve. Then he began to have a rather septic look. As the jugular had been tied below the entrance of the facial vein it was felt that there could be no danger from that source. There was no purulent process on the left side. Very slowly, but surely, he was losing ground. A few times he complained of headache. On the seventh day there was some stiffness of the neck; the eye-grounds showed no changes. Kernig's sign was absent. There were leukocytosis of 13,000; polynuclear percentage of 83; and streptococci.

Knowing that a frequent source of infection of the cerebellum is through the sinus it was decided to explore, even though there were no definite symptoms. The route chosen was through the sinus. A small cerebellar abscess was found and drained. Again for a few days there was apparent improvement, but then he began to lose ground and on August 18, not quite six weeks after admission, he died. The autopsy showed that the jugular, the sinus, and the cerebellum had healed completely, but that there were multiple abscesses of both lungs, the liver, and the spleen.

It seems to me that the diagnosis of these conditions is the most difficult part of our work. No group of symptoms will be found infallible and no symptom is pathognomonic. Any one of the symptoms may be present or absent, with possibly one exception. I have come to place great reliance upon the presence of leukocytosis with high polymorphonuclear percentage. If this is 80 or over it has always, in my experience, shown sepsis; not necessarily of the brain.

It may be septic thrombosis or other general septic process, but in my experience its presence has never yet failed to indicate sepsis. Its absence does not contra-indicate sepsis, as shown in cases that have been reported in which even with a high degree of pyemia the polynuclear percentage may be as low as 70. But given a group of symptoms pointing to an infection, the presence of a high polynuclear percentage will, in most cases, be the deciding factor.

REVIEWS.

Modern Medicine. Its Theory and Practice. In Original Contributions by American and Foreign Authors. Edited by William Osler, M.D., Regius Professor of Medicine in Oxford University, England. Assisted by Thomas McCrae, M.D., Associate Professor of Medicine and Clinical Therapeutics in Johns Hopkins University, Baltimore. In seven octavo volumes; illustrated. Vol. I, pp. 937. Philadelphia and New York: Lea Brothers & Co., 1907.

The publication of Osler's *Modern Medicine* must be considered an event in the history of medicine in this country; it has been awaited with keen anticipation, and the conviction has been general that it is destined, even more than systems of medicine heretofore published, to influence profoundly medical thought and practice in the English-speaking world and beyond. The purpose in mind and the object of the work are well stated by Dr. Osler in his introduction, when he says: "It is designed primarily for the practitioner who wishes to keep himself informed of the existing state of our knowledge in clinical medicine. Elaborate discussions upon doubtful problems have been avoided, and, as far as possible, a clear statement is given without unnecessary references to the literature. Authors have been selected who are acknowledged authorities, and while it is not always easy for a writer who is saturated, so to speak, with his subject to keep within limits, and to remember the practical character of the men for whom he is writing, I hope we have been able to keep an even balance between the condensation of the text-book and the elaborate treatment of the monograph. The first consideration in a work of this kind is that it should be helpful."

In addition to the introduction by Dr. Osler, the volume contains the following articles: Inheritance and disease, by J. George Adami; diseases caused by physical agents (light, *x*-rays, electricity, air, heat, and cold), by Alfred Gordon; diseases caused by chemical agents (metallic and other poisons), by David L. Edsall; drug habits (alcohol, opium, morphine, cocaine, etc.), by Alexander Lambert; food poisons, by Frederick Novy; snake poisoning, by Hideyo Noguchi; auto-intoxications, by Alonzo Englebert Taylor; actinomycosis, nocardiosis, mycetoma, oidiomycosis, blastomycosis, pulmonary aspergillosis, and mycosis mucornia, by James H. Wright;

the protozoa, by Gary N. Calkins; mosquitoes, by L. O. Howard; malarial fevers, by Charles F. Craig; black-water fever, by J. W. W. Stephens; trypanosomiasis, by David Bruce; amœbic dysentery, by Richard P. Strong; diseases caused by animal parasites, by Charles W. Stiles; metabolism, normal and in disease, by Russell H. Chittenden and Lafayette B. Mendel; diabetes mellitus, diabetes insipidus, and gout, by Thomas B. Futcher; obesity, by James M. Anders; rickets, by George Frederic Still; and scurvy, by Robert Hutchison.

The range of subjects discussed is so wide and diversified that it is impossible within the limits of a reasonable review to do more than mention the titles of many of the contributions: one must content himself with generalities, rather than undertake a critical review. Most of the contributors have a national, some an international reputation, and their names alone constitute assurance of excellence of accomplished work; indeed, so well known is the association of the author with his work that were those competent to judge asked to designate authorities preëminently fitted to contribute certain chapters, they doubtless would mention Adami, Calkins, Howard, Craig, Stephens, Bruce, Strong, Stiles, Chittenden, and others of the afore-mentioned list.

If the reviewer ventures to select for special comment several of the many articles in the volume, he does so conscious of the excellence of those referred to only casually. Thus, it seems a happy thought that suggested the incorporation of articles on the protozoa and the mosquitoes—articles, at first sight, foreign in scope and nature to a work on medicine. However, although we have scarcely more than begun to comprehend the pathogenetic significance of the many protozoa, we do know that a knowledge of the varieties, structure, and life-history of the mosquitoes is essential in the study and prevention of malaria, yellow fever, and other diseases; so that upon reflection it seems exceedingly appropriate that these chapters, written by acknowledged authorities, should be included; furthermore, they give to the practitioner information nowhere so readily obtainable and nowhere presented in a form so well adapted to his needs.

Several of the articles must be looked upon as really noteworthy contributions to medical literature. Of these, one may perhaps mention first those by Adami, Taylor, and Chittenden and Mendel. During the last twenty years our ideas of heredity in relation to disease have undergone considerable modification in consequence of the studies of Weismann, Mendel, and others, and it is meet, therefore, that we should have such a clear and comprehensive, though withal succinct and authoritative, statement of present opinions on the subject, as Adami gives us. He well separates conditions mistakenly regarded as inherited from those truly inherited. Especially illuminating is his discussion of individual inheritance and the theories of inheritance, and his exposition of his own conception of a physicochemical theory of inheritance. Taylor's article on auto-intoxication is unquestionably one of the sanest discussions of a subject which so readily lends itself to speculation that we have read during recent years. Particularly commendable is his treatment of gastro-intestinal auto-intoxication, of which he adopts an exceedingly sensible classification—satisfactory alike to the scientist and the practitioner. One minor regret is that the clinical phenomena of the condition have not been more elaborated; but the article as it stands is commended to the attention of all who desire to separate fact from fancy in the realm of auto-intoxication. The discussion of metabolism, normal and in disease, by Chittenden and Mendel, will appear to the scientific physician as the final pronouncement of well-qualified investigators; by the practitioner it will be read with interest if for no other reason than that it runs counter to some of his preconceived ideas in reference to food values. It contains also the results of considerable investigation that may be used by the physician in formulating rules for the dietetic management of disease. This matter of diet is of very great importance, and no investigations have aroused greater interest than those recently conducted by Yale University. If they seem to prove that most persons eat too much, especially too much meat, there is, on the other hand, a large number of competent observers who do not accept the result of these investigations *in toto*, who believe that although certain things may be possible to the human economy, these may not be wise, and that withdrawal of much proteid may occasion undesirable demands upon the reserve power of the system, especially as regards non-proteid metabolism. A sane view of the matter, however, is thus well expressed by Chittenden and Mendel: "One is inclined to emphasize the desirability of using common sense in the application of dietetic rules, remembering that man is well adapted by nature to subsist on a variety of foods. On matters of diet every man should be a law unto himself, using judgment and knowledge to the best of his ability, reinforced by his own personal experiences. Vegetarianism may have its virtues, as too great indulgence in flesh foods may have its serious side; but there would seem to be no sound physiological reason for the complete exclusion of any one class of foodstuffs, under ordinary conditions of life."

In addition to the foregoing, the volume contains a number of articles of much practical and other value. The chapters on diseases due to chemical agents (metallic and other poisons), by Edsall, is of a very high order of merit; and it gives evidence of a first-hand acquaintance with the disorders in question: notably in connection with chronic mercurial poisoning, but also elsewhere. The articles on diabetes mellitus, diabetes insipidus, and gout, by Futcher, are such as a knowledge of his earlier work would lead one to expect: complete, authoritative, practical. Lack of space only forbids a more detailed mention of the other really excellent articles.

As a whole the volume is a noteworthy addition to medical literature—well conceived, excellently planned, splendidly executed. It reflects, doubtless as he wished, the thoughts the editor had in mind when he wrote: "It cannot be too often or too forcibly brought home to us that the hope of the profession is with the men who do its daily work in general practice. Our labors are in vain—all the manifold contributions of science, the incessant researches into the complex problems of life, normal and perverted, the profound and far-reaching conclusions of the thinkers and originators—all these are *Nehushtan,* sounding brass and tinkling cymbals, unless they result in making men better able to fight the battle against disease, better equipped for their ministry of healing. Gradually, often insensibly, the practical advances of the laboratory and of the hospital reach the men with whom, after all, rests the final testing of our efforts. The work in practical sanitation, the last word in the prevention of disease, the carrying out new methods of treatment, the exchange of the old accoutrements for the new weapons and the new methods of warfare, these rest with the rank and file of the profession who make effective and translate into practice the new knowledge."

Volume I being a foretaste of what is to come, Osler's *Modern Medicine* may be warmly recommended to the physician as replete with the practical applications of science to medicine, mirroring the clinical manifestations of disease, and helpful and resourceful in suggestions for treatment. A. K.

A STUDY OF HUMAN BLOODVESSELS IN HEALTH AND DISEASE. By ARTHUR V. MEIGS, M.D., Physician to the Pennsylvania Hospital, Philadelphia. Pp. 136, with 103 original illustrations. Philadelphia and London: J. B. Lippincott Co., 1907.

THE reader who takes up this volume with the expectation of finding it a systematic treatise upon vascular disease will be disappointed. It is a book of personal observations and interpretations, and nowise a compendious or systematic presentation of the pathological changes to which bloodvessels are liable and which they frequently exhibit. The book is based upon a series of excellently executed illustrations made by the eminent medical artists, the Fabers, from microscopic preparations studied by Dr. Meigs; and in fact should be considered as an atlas, with the text mainly made up of explanatory descriptions of the drawings and entering but little into further analysis and discussion than such description entails or into attempt at correlation of the author's findings with the work of other students. The bulk of the preparations have been made with borax carmine as the stain; and from the outstart the author, therefore, largely limits his scope to features which this reagent yields, and misses much really valuable information which some of the more differentiating special methods of staining would have revealed. The text includes chapters upon the structure of arteries, veins and capillaries, new bloodvessels, diseases of bloodvessels in general, diseases of the radial arteries and veins, bloodvessels in inflammation, tuberculosis and syphilis, the bloodvessels of the heart, lungs, liver, spleen, kidney, and of the brain and spinal cord. That the mode of presentation has opened the way for unnecessary and undesirable repetition of statement, into which unfortunately the author has fallen, is an entirely fair criticism; and that for the comprehensiveness of the title and the ambitious appearance of the volume the treatment is too discursive and the isolated subjects in too unfinished a state seems to the reviewer an equally just comment. The author's views would have gained added force had he grouped his material and illustrations upon this or that type of vascular disease instead of distributing, without actual addition, his statements and examples over the general and various special chapters; at any rate such method would have ensured a greater economy of pages and would have saved the author the self-confessed imperfections of most of his special chapters.

These strictures are presented in good faith and with no intentional carping criticism. It is realized full well that the volume was not published as a text-book or treatise but rather as a record of observations made as a life heavy with the duties of the active practice of medicine have permitted; yet if in future editions the method of arrangement could be altered by condensation and subject rearrangement, and if a closer correlation and fuller study of some of the more interesting and important features could be attempted, the book would surely gain much in excellence.

As it stands there is contained in the volume much food for thought; and the acuteness of the author's power of observation and the originality of his interpretations make it a very suggestive book and one well worth perusal. In the first chapter the demonstration that the artefacts due to shrinkage of vessels in fixation and preservation may be responsible for mistaken recognition of various irregularities in the walls of both arteries and veins, but more markedly in the latter, is very successful; and differences of appearance given in the coats of vessels into which suitable glass rods or tubes have been inserted before fixation in comparison with those afforded by vessels ordinarily prepared, are striking and worthy of remembrance. Minor thickenings of the intima are clearly shown to be possibly nothing more than such artefacts and not necessarily to be regarded as evidence of disease. The observations upon the structure of the vena cava and the cardiac sinus venosus deserve close attention (absence of circular muscle layer from the wall of the vena cava; lack of differentiation into the usual coats, and absence of smooth muscle from the wall of the sinus venosus, which consists merely of a thin fibrous intima merging by a loose fibrous tissue directly with the tissue of the myocardium). In the chapter upon capillaries the author proposes a theory of the evanescent character of these tubes which the reviewer feels is very unlikely to meet with any ready approval or acceptance. He compares the capillaries with the little rills of water which form between blades of grass after a heavy rain, trickling forward to join in greater and greater volume until poured into some permanent stream, but disappearing as the ground surface dries. "They may be truly said to be ephemeral, for they open in the tissues as the need for them arises and close and disappear when their usefulness has ceased. . . . It is certain that capillaries are ephemeral and often return to their primitive state by giving back their walls again to form simple connective tissue. . . . The capillaries, on the other hand, are nothing but tubes hollowed out in the connective tissue with which they are surrounded. If a single capillary running in any tissue by itself is empty it becomes at once and without any change a string or fiber of connective tissue." (Pages 17 and 19.) Undoubtedly with varying grades of fulness the capillaries vary in ease of recognition, but the reviewer is entirely unwilling to subscribe to the author's statement of the complete conversion of their endothelial structure into strings or fibers of connective tissue and the reassumption of the plate-like character of their cells with alternate grades of emptiness and fulness of the tubes. Examination of the largely bloodless and shrunken tissues of a microscopic preparation of autopsy material is far apart from examination of living structures; and observation of such preparations as a frog's mesentery, foot-web, or tongue in the living animal before inflammatory changes have set in, give an impression of capillary fulness and ubiquity in the tissues very much at variance with the author's fancy. That under pathological conditions with due time capillaries may form anew and are often also in time destroyed and contribute to connective tissue goes without saying at the present day; but were it a serious question the proof of the incorrectness of Dr. Meigs' surmise would not be difficult of demonstration. His own criticism of the old idea of the incompleteness of the capillary arrangement in the spleen, necessitating a diffusion of blood in splenic pulp (p. 114), if applied to other structures of the body can with difficulty be harmonized with such ideas. In his chapter on new bloodvessels there is much of interest in the suggested formation of capillaries in the manner of vessel development in the embryonic vascular area. This mode of vascular production in pathological processes has never been successfully made out; but the observation of capillary growth within a tissue, which to the reviewer's mind is hyperplastic endothelium, is at least suggestive, not so much in the mere formation of the vessels, since it is quite possible that they may have been the usual capillary penetrations from the nearby vasa vasorum, but in the features which the author interprets as evidence of their formation independently of any other vessels. Should Dr. Meigs' views come to prevail in this matter alone, after due study and confirmation, his whole volume would be well justified. In this same chapter the magnificent etching of giant cells (Fig. 25) is an admirable argument for the belief in the vascular origin of these structures in tubercles; and the author's view that they represent abortive attempts at vascular formation, while not established by his demonstration, is surely worth serious consideration and study. Probably the best chapters in the volume are those upon disease of bloodvessels in general and upon disease of veins; and of the particular features of these parts the best is the clear demonstration of the frequent and important involvement of veins by sclerotic disease, and the more ready invasion of venous than arterial walls by acute inflammatory infiltrations. Here, as well as elsewhere in the volume, the demonstration of well-marked sclerotic changes in the vessels of the young, notably in several children, is a feature of decided value. Of the special chapters, as might be supposed from the author's previous writings, that upon the bloodvessels of the heart, repeating his well-known demonstration of the intramuscular as well as intermuscular situation of the myocardial capillaries, and demonstrating them by injection preparations in addition to his previous studies on non-injected preparations, is the most satisfactory.

All through the volume occur expressions and views with which one could if so disposed take quick exception, for example, the author's very evident lack of sympathy and neglect of modern views of the importance of microbic influence in the origin of disease (p. 99) or his refusal to recognize anything morphologically peculiar in the nodular groupings of cells in tubercle formation (p. 69); but the reviewer prefers to avoid the really needless discussions which would be entailed by their consideration. The pictures upon which the book is based are very valuable, and are presented with an enviable luxury, being printed upon inserted sheets of a well-calendered paper and accompanied with full legends upon the opposed tissue cover. The reviewer has noted only one typographical error (p. 81, line 10, substitute "recognized" for "reorganized"); and the whole mechanical work is good. A. J. S.

A Text-book on Prescription Writing and Pharmacy. By Bernard Fantus, M.D., Professor of Materia Medica and Therapeutics, College of Physicians and Surgeons, University of Illinois, Chicago. Second edition, pp. 404. Chicago: Chicago Medical Book Co., 1906.

If one may select that in which this book appeals to him above the rest, he would state that in making every endeavor that the student shall become proficient in prescription writing, the author has achieved remarkable success: not only that prescriptions shall be in unimpeachable terminology, but that the student shall originate prescriptions adopted to the requirements in the special instance of disease and the particular patient suffering from it. Not only in calling attention to the externals, those small matters which indicate the man of science, but in citing especial instances of bad form, the author presents the questions of determination of quantities, the composition of prescriptions, covering also modes of administration of remedies, and closes his first part with sensible suggestions in prescription ethics. Part II, Pharmacy, covers the various classes of preparations, with frequent examples of what the student will designate as "good prescriptions." Part III is a reference list of official drugs and their preparations, and contains much information in small space which the student will find useful in formulating his therapeutic endeavors; for instance, the name with indicated pronunciation, the genitive, the definition, description, solubility, incompatibles, uses in brief, and method of administration. The student who acquires this will learn not only how to prescribe elegantly, but as well will acquire considerable pharmacological and therapeutic information. In the appendix is found a few suggestions for laboratory exercises. These we regard as essential and if carefully taught with blackboard exercises in writing, the future physician will appear in a higher scientific position than the pharmacist has given him credit for. We would especially commend this work to those compilers of inspired therapeutic literature based on selected formulæ of eminent physicians so frequently met with in trade circles. We feel that a study of this book will conduce not only to more elegant, but as well to more effective therapeutics.

R. W. W.

PARAFFIN IN SURGERY: A CRITICAL AND CLINICAL STUDY. By WILLIAM H. LUCKETT, B.S., M.D., Attending Surgeon, Harlem Hospital, and Surgeon to Mount Sinai Hospital Dispensary; and FRANK I. HORN, M.D., Assistant Surgeon, Mount Sinai Hospital Dispensary, New York City. New York: Surgery Publishing Co., 1907.

THIS little monograph presents in a concise form a good deal of original work as regards the use of paraffin in surgery. Its authors present successively the subjects of the chemistry of paraffin, its early and late disposition in the tissues, the technique and armamentarium required for its injection, and the unsatisfactory results and accidents which occur from its use. As to its disposition in the tissues, they hold, as a result of microscopic sections made immediately after its experimental injection into the tissues, that the early disposition of paraffin is a mechanical and physical distribution in the tissues. They believe that they have furnished conclusive proof of the disinclination of pioneer cells, fibers, and bloodvessels to permeate the mass of paraffin. Their experiments also show that paraffin in the tissues becomes encapsulated, consisting of new tissues formed from tissues encircled by paraffin at the time of the injection; accordingly encapsulation takes place from the inside as well as from the outside. They consider that the accidents which have occurred, such as amaurosis and pulmonary embolus, are attributable to the use of paraffin with too low a melting point. They recommend the use of paraffin at a melting point above 110° F. The last section of the book is occupied with reports of cases in which they have used paraffin injections. Whereas of late the pendulum is swinging violently in the direction of too great depreciation of the value of paraffin in surgery, the authors' efforts to throw some real light on the subject are most commendable, and their conclusions show that, if properly utilized, paraffin is a most valuable therapeutic agent. F. R. P.

THE EAR AND ITS DISEASES. A TEXT-BOOK FOR STUDENTS AND PHYSICIANS. BY SETH SCOTT BISHOP, B.S., M.D., LL.D., Honorary President of the Faculty and Professor in the Post-graduate School and Hospital of Chicago. 27 colored lithographs and 200 additional illustrations; pp. 440. Philadelphia: F. A. Davis Co., 1907.

THIS work is an amplification of the section on the ear which was included in the author's previously published book on *Diseases of the Nose, Throat, and Ear.* Dr. Bishop deserves to be heartily congratulated upon the result of his labors. In the present volume he has covered the entire subject of otology in the most comprehensive manner, and as his literary style is concise and lucid, within the upward of four hundred pages which were at his disposal, the reader may search in vain for sins of omission.

The opening chapters on the anatomy of the ear are very fully illustrated, chiefly with original drawings from the author's specimens. In fact, the illustrations are a noteworthy feature throughout the entire work; the only criticism to be made in regard to them is that there are several which might be deemed superfluous, such as the interior of an office and of an operating room. Dr. Bishop has devised an excellent case record, one of the most practical we have ever seen. All of the topics written of are thoroughly well handled. This is particularly true of the section dealing with chronic adhesive middle-ear inflammation. The author advances more ingenious and practical methods of treatment for this *bête noir* of the aurist than are usually described, and shows an amount of common sense and judgment that is too often wanting when various plans of treatment are under consideration. He is properly emphatic in warning against injudicious overtreatment of the sclerosed ear.

We do not know when we have reviewed a book written on otology and meant for the use of students and general practitioners, as well as specialists, in which so much of real value to all of the three groups mentioned is combined. Dr. Bishop's book is admirably adapted for use as a text-book and as a book of reference.

F. R. P.

Metabolism in Practical Medicine. By Carl von Noorden. Vol. I, The Physiology of Metabolism. By Adolf Magnus-Levy. Anglo-American issue, under the editorship of I. Walker Hall. Chicago: W. T. Keener & Co., 1907.

One very commonly hears expressed a desire for an authoritative, philosophical discussion, in a manner that can be appreciated by the intelligent general practitioner, of the results of the vast amount of work that has been done in comparatively recent years on metabolism. The studies of this subject have been so very numerous, and have, in large part, been carried out by such relatively technical methods, that many intelligent practitioners have only a hazy idea of the actual value of the results; and, in ordinary circumstances, it is practically impossible for them to clear up their conceptions without undertaking a very laborious study of the literature of the subject. The work of which this is the first of three volumes in large part fills this want. Von Noorden's original *Pathologie des Stoffwechsels* was of great value to the special student of metabolism, but not very interesting to the clinician who had no such special interest: because it was largely a concise statement of figures and conclusions. In this present new and greatly elaborated edition of this work he has associated with himself a group of the most important of the workers on metabolism in Germany, who have taken up subjects concerning which they speak with authority; and the questions are discussed from a much broader and more philosophical standpoint, and in a manner that it is both interesting and comprehensible to the practitioners in general.

The only unfortunate thing about the work is its size, which will, both from the standpoint of cost and from that of the labor involved in going through it, interfere to a considerable extent with its being widely read. Those, however, who have the moderate amount of energy demanded to read it will find it a most valuable and interesting discussion of all the important questions concerned in metabolism, so far as our knowledge at present goes. The only point to which serious exception can be taken is, perhaps, that the authors limit themselves rather too much to a consideration of German literature, largely to the exclusion of work done outside of Germany; but, this being a common custom with German authors, it deserves no special emphasis here.

The reviewer's acquaintance with the German edition of this work leads him to feel that the present volume is the most valuable to practitioners of medicine, because it especially well gives them increased breadth of view; the other volumes dealing with more detailed subjects. The author of this volume, Magnus-Levy, as would be expected from his various writings, takes a broad, firm, and interesting point of view in his manner of discussing the subject; and gives fair credit to all sides of any contentions. He takes up the foodstuffs, digestion and absorption, the fate of the foodstuffs in the tissues, total energy exchange, nitrogenous metabolism, the influence of muscular work, and various other such factors. It is an exceedingly valuable work and the English-speaking profession should be grateful to have a translation of it. D. L. E.

ALCOHOL. THE SANCTION FOR ITS USE. Translated from the German of J. STARKE. New York and London: G. P. Putnam & Sons, 1907.

POPULAR and semipopular books on important scientific subjects are, as a rule, of questionable utility; and they are likely to be very harmful, if they are so prepared that they readily lead persons astray. The common statements regarding alcohol in the text-books on physiology used in the public schools are, as a rule, both ludicrous and harmful. These statements are usually violently against alcohol. The book at present under review takes the contrary standpoint, and gives a most absurd discussion of the value of alcohol to humankind; and it is likely to do a good deal of damage, because it is written for the laity.

From the scientific standpoint, a review of the book is superfluous. There are many details that are extremely entertaining, because they are so funny; but the fairest method is perhaps to close the review by quoting a sentence that the author introduces after a somewhat involved discussion of the difference between a "weak character" and a "weak mind." "In short, and the reader will agree with me, it is not prudent to go into too much detail in this matter."

D. L. E.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Mouth Disinfection in the Prophylaxis and Treatment of Pneumonia.—WADSWORTH (*Jour. Infect. Diseases*, 1906, iii, 774) has studied the effect of disinfection of the mouth in the prophylaxis and treatment of pneumonia, his studies comprising cultural, morphological, and animal experimentation, and the effects of simple isotonic salt solutions, alcohol, different bland solutions, and various commercial solutions. Among his conclusions the following points may be noted: His own observations confirm the results of previous studies that complete disinfection of the mouth is impossible, but from tests and experiments the practical value of many of the antiseptic procedures commonly employed in mouth disinfection was accurately determined. Pneumococci, as compared with other pathogenic bacteria, are extremely susceptible to the plasmolytic action of saliva and alkaline solutions and also to the antiseptics which act in this way. The susceptibility to antiseptics in general varies greatly and especially under different conditions. In broth media pneumococci are readily destroyed; in exudates, albuminous material and detritus interfere with the action of many antiseptics. Sputum disinfection by harmless solutions is extremely difficult. Of all the commercial solutions studied—listerine, borine, borolyptol, glycothymoline, odol, and Seiler's solution—none proved efficient when tested on pneumococci under conditions most favorable for their action. Formalin, lysol, and hydrogen peroxide failed to act on the pneumococcus in exudates; in short, alcohol alone of all the antiseptics studied proved efficient when tested on pneumococci under all the conditions of the experiments. Observations on the diffusion of different solutions in the secretions of the mouth under different conditions showed that the presence of salts in isotonic quantities and alkalies was of positive, if slight, value. Rapid diffusion obtained with alcoholic solution was greatly accelerated by the addition of glycerin. Hot solutions are better. In the practical studies on pneumonia patients, simple isotonic solutions are only slightly efficient; but with an alcoholic wash many of the bacteria are destroyed, the contaminated secretions are more rapidly removed, and at the same time disinfected, and finally the natural resources of the tissues are more safely and efficiently aided in the elimination of the infectious material. Thus, alcoholic solutions containing glycerin and salts in bland quantities were in every particular more efficient than any of the washes hitherto recommended for mouth cleansing or disinfection and alone possess practical value. 30 per cent. of alcohol is the strongest solution that can be used habitually, and the weakest to give for reliable disinfection. This disinfection should be urged most emphatically on each individual, owing to the ease of spreading the infection by the transfer of the infectious material by coughing or sneezing, especially when in crowded communities. The formula Wadsworth found most suitable was as follows:

℞—Sodium chloride. (C.P.)	2.0	½ dram.
Sodium bicarbonate. (C.P.)	0.7	10 grains.
Distilled water	60.0	2 ounces.
Glycerin	30.0	1 ounce.
Alcohol	150.0	5 ounces.
Thymol Menthol, of each	0.07	1 grain.
Oil of wintergreen		3 drops.
Oil of cinnamon		2 drops.
Oil of eucalyptus		5 drops.
Tincture of cudbear	6.0	1½ drams.
Tincture of rhatany	2.0	½ dram.—M.

S.—Dilute with an equal volume of water before using.

The Opsonic Index and the Antipneumococcal Power of the Blood in Pneumonia.—H. E. Wolf (*Jour. Infect. Diseases*, 1906, iii, 731) gives a brief summary of the work of Wright and Douglas, Mennes, Hektoen, and Neufeld and Rimpau and their results obtained in conditions other than pneumonia. He experimented with different dilutions of normal and pneumococcal serum and his method of work is outlined It was found that dilutions of 0.025 c.c. gave the most satisfactory results; the pneumococco-opsonic indices in the charts are based on results so obtained. 14 cases were studied by this method, usually with daily determinations, before the crisis in typical cases, and less frequently afterward. 2 cases were complicated with empyema, 2 were of a migratory type, and 5 were typical lobar pneumonias ending with crises. Besides the opsonic index, leukocyte counts and blood cultures were made, and are indicated in the interesting charts for comparison. The typical cases had much in common. The opsonic index persisted below normal until within a few hours before the crisis (this corresponds to the negative phase of Wright), then rose and reached a maximum within twenty-four hours after the crisis; then followed a slow decline to normal. During the same time the leukocyte curve rose with the chill and temperature and began to decline as the opsonic index rose; thus although the individual pneumococco-opsonic index of pneumonic blood may be below normal early in the disease, yet due to the increase in the leukocytes, the antipneumococcal power of the same may be much above the normal, assuming, of course, that the opsonin present is sufficient to opsonize the quota of pneumococci necessary. Rosenow's observations (*Jour. Infect. Diseases,* 1906, iii, 683) on the pneumococcidal action of polymorphonuclear leukocytes *in vitro* support this view. This is shown very graphically in one of the charts. In one of the cases of migratory pneumonia, the pneumococco-opsonic index remained persistently below normal, rising a little with the clearing up in one lobe, but falling the next day when the other lobes were involved. At last on the twelfth day they rose above normal and continued so throughout the period of delayed resolution until complete recovery.

In the fatal cases examined the opsonic index continued to fall until death. The leukocytes, though high, fell steadily during this period. Two interesting experiments were performed on normal, healthy men. In one the injection of dead, virulent pneumococci gave some local inflammatory signs with an increase in temperature and leukocytes, with at first a drop in the opsonic index (negative phase) and then a rapid rise above normal with a coincident drop in the number of leukocytes. In a second experiment with dead avirulent pneumococci, somewhat similar effects were noted in the temperature and leukocytosis, but there was no increase in the opsonic index. This indicated that the property of virulence may be somewhat concerned in the stimulating of the production of immune opsonins.

Wolf then notes the effects of injection of pneumococcal opsonins in 14 cases; 11 recovered, 3 were fatal. In all cases ending favorably there was a slight increase in temperature four to six hours after injection and an increased leukocytosis. Crisis occurred in from eighteen to thirty-six hours after injection in all but one case, in which a second injection was given and crisis followed in eighteen hours. The opsonic index rises characteristically just before the crisis. There was no exaggeration of the negative phase immediately following the injections. In 5 cases the crisis occurred on the third day, 3 on the fourth, 1 on the fifth, and 2 on the sixth. These cases were chosen at random from different patients, with a mortality of about 40 per cent. for that month in the hospital. Hence the recovery among the infected patients seemed to be high and crisis occurred early. Of the 3 fatal cases 2 were senile pneumonias, while the others showed a marked hypoleukocytosis. After injection there was no marked reaction in the temperature or leukocytes and no change in the opsonic index, possibly indicating that the sources of vital resistance of the body had been so exhausted as to permit of no further stimulation. His conclusions are as follows: (1) In pneumonia the pneumococco-opsonic index is first decreased, but it rises in favorable cases, reaching its height soon after the crisis, while in unfavorable cases it remains persistently low. (2) The estimated total antipneumococcal index (estimated from the leukocytic index and opsonic index) is early increased and remains high until crisis is complete in cases with a favorable termination. (3) Pneumococcal exudates contain little or no pneumococco-opsonin, nor do they exert any antiphagocytic action. (4) Dead virulent pneumococci produce a similar evolution in the antipneumococcal index in normal persons to that which occurs in pneumonia. (5) Dead avirulent pneumococci have no effect on the pneumococco-opsonins. (6) Treating patients with lobar pneumonia with antipneumococcal vaccines seems to exert a favorable influence upon the course of the disease, as indicated by an apparent decrease in mortality and by early crisis, but a great deal of further work is necessary before any conclusions of final value may be drawn.

The Functions of the Muscles and Nerves of the Arteries. GRUTZNER (*Deutsch. Arch. f. klin. Med.*, 1906, lxxxix, 132). The earlier work of Claude Bernard, Ludwig, and Heidenhain established the existence of dilator and constrictor fibers to the arterial muscles and showed the importance of their action in maintaining and regulating local and general blood pressure. The question arises whether this stop-cock action of the arterial musculature is its only function. Senac was the first to suggest that the arteries through their muscles serve not only to widen and constrict the flow-bed, but take an active part in the propulsion of the blood. "The arteries," he says, "are real hearts of a different form." This view was strengthened by the observation of Schiff that the arteries in the rabbit's ear may be seen to contract in an orderly sequence from base to tip. Many facts have been adduced to sustain the existence of such a propelling force in the arteries. That the arteries are empty after death and the veins filled is one. It has been observed that the circulation continues in the frog's foot for some time after the heart is tied off. Bezold and Goldscheider observed that after tying off the heart in rabbits the pressure in the carotid and in the vena cava were about the same, namely, about 86 mm. of water. If the cervical cord were severed and the vasomotor nerves thus cut off the arterial pressure was 86.6, the venous 43.1. If the vasomotors in the severed cord be stimulated the venous pressure will rise; for example, from 50 mm. to 75 mm., while the arterial pressure falls. Grützner also points out that when sensory nerves are stimulated there is a rise of blood pressure which Ludwig explains by a constriction of the splanchnic area, but the same rise occurs when the whole splanchnic area has been ligated off. Such a procedure should cause a dilatation of the muscle and skin vessels, and unless we assume that the arteries themselves have some propulsive power it is difficult to understand how the pressure is increased. The distribution of the muscle fibers, too, is an indication of their purpose. They are most numerous in the small arteries where the force of the heart becomes spent, and particularly abundant in places where special difficulties must be overcome, as in the intestinal capillaries. The strong musculature of the vena porta and of the umbilical artery indicates a response to the demand where most assistance is needed to carry on the circulation. The capillaries possess no muscles, but they may dilate and contract by means of the surrounding Rouget-Meyer Korpzellen. The veins have muscles and no doubt assist in propelling the blood. Goltz has shown that paralysis of the abdominal veins renders the circulation impossible, the animal bleeding to death into its own veins. The venous sacs with rhythmic contractions in some of the lower animals offer suggestive analogies. From the clinical side similar conceptions of the importance of the vessels in assisting the heart action and in controlling the distribution of the blood have come from Rosenbach and Hasebroek.

Diseases of the Large Bloodvessels in Congenital Lues.—Rach and Wiesner (*Wien. klin. Woch.*, 1907, xx, 521), in an examination of 27 cases of severe and mild congenital lues, found lesions of the aorta or pulmonary artery in 16. The lesions are most frequently situated just beyond the point of origin of the aorta and at the orifices of the large arteries, at which points the vasa vasorum are most numerous. They consist histologically of an infiltration with small and large lymphocytes and a few polymorphonuclear leukocytes and proliferation of the connective-tissue cells of the adventitia and neighboring layers of the media. The changes are most marked immediately about the vasa vasorum and frequently lead to obliteration of their lumen. In some instances there is degeneration of the elastic fibers of the media. In the descending aorta of one case an elevated plaque was found on the intima consisting of fibrillary connective tissue poor in cells. The elastic lamellæ were separated and many of their fibers destroyed. Such changes in the intima and media are rare. The lesions in the bloodvessels bear no relation to luetic disease of the other organs. The one may be severe while the other is slight. Careful search failed to reveal spirochetæ in any of the lesions and the authors are inclined to view them as toxic in origin. The frequency with which the arteries are affected in congenital lues suggests an etiological factor for many cases of arteriosclerosis of unknown origin in children. Such early lesions may too be a predisposing factor in the development of arterial changes in later years.

The Causes of Orthopnœa.—Hofbauer (*Ztschr. f. klin. Med.*, 1907, lxi, 389) takes exception to the prevalent view that patients with dyspnœa assume an upright posture because this position facilitates the action of the accessory muscles of inspiration and relieves the chest from the pressure of the body weight. Such a simple explanation does not satisfactorily account for the increased difficulty of breathing which so commonly comes on during the night. Some other factor must be at work. In cases of pleural disease Hofbauer has shown by carefully studied tracings that the dyspnœa is expiratory in type, the difficulty being in getting the air out of the lung and not in getting it in. The dyspnœa depends upon loss of elasticity in the lung, the retraction energy being compromised so that the expiratory phase which normally is passive must be assisted by muscular effort. During the day the patient throws the necessary muscles into play subconsciously, but during sleep the cortical centres fail to supply the necessary stimulation and this accounts for the frequency with which the patient is awakened from sleep in an attack of dyspnœa. The difficulty of expiring being the cause of the dyspnœa, expiration is evidently facilitated by the upright position because this is the posture that gives most relief to the patient. Hofbauer has studied the mechanism of this relief by careful *x*-ray examinations of the position of the diaphragm in various postures. Very unexpectedly he has found that the diaphragm is higher in position and its excursions far wider in the lying than in the sitting or standing position. This apparently contradictory finding Hofbauer explains thus: The lung has an adherent tendency to contract, and whenever the negative pressure in the pleural cavity is diminished, as by an accumulation of air or water, the lung assumes gradually the contracted state. This tendency to contract is one of the chief factors in the expiratory phase, and the more distended the lung the more forcible will be the subsequent contractions. In pneumothorax, for instance, the dyspnœa is caused by the drawing of the mediastinum over into the unaffected side, thus hampering the dilatation of the normal lung. At operation, if the mediastinum be seized with forceps and drawn into the normal position, the dyspnœa stops at once. In a sitting posture similar conditions obtain. The low position of the diaphragm increases the capacity of the chest and holds the lung in a more distended condition so that the elastic expiratory recoil becomes more efficient. The dyspnœa being expiratory in type this renders breathing easier.

Tabes Dorsalis, Disease of the Circulatory Organs, and Syphilis.—Rogge and Miller (*Arch. f. klin. Med.*, 1907, lxxxix, 514). Although the association of tabes and disease of the circulatory organs, especially of aortic insufficiency and aneurysm, is well known, the frequency of the combination and its clinical importance has not been sufficiently emphasized. In 22 consecutive cases entering von Strumpell's clinic 8 showed definite signs of cardiac or arterial disease. This is probably an unusually high percentage, and the examination of the total clinical material gives the occurrence of definite organic heart or aortic disease as 10 per cent. Such a frequent association indicates that a common causative factor underlies both. In 24 selected cases in which the two diseases occur together Rogge and Miller find that disease of the aortic orifice is by far the most common complication, occurring in 15 instances. Aneurysm was present in 9 instances; never isolated, but in combination with other serious disease of the arterial system and most frequently with aortic insufficiency. The aneurysms were all fusiform, resembling the marked dilatation of the arch so commonly found in combination with the aortic insufficiency following arteriosclerosis. In several instances there were marked clinical signs of a pure myocarditis, and in others advanced arteriosclerosis at an early adult age—twenty-seven to thirty-five years. It is interesting to note that subjective symptoms were complained of in only about half the cases, the cardiac disease being discovered in the course of a routine examination. This suggests that many of the sudden deaths in tabes may be more closely related to disease of the circulatory apparatus than to degeneration of the bulbar centres. It emphasizes, too, how frequently the severe pains usually ascribed to purely nervous influences may have a real anatomical basis. Syphilis is the most important factor in the etiology, 9 cases giving a definite history and 10 more a very probable history. Other infectious diseases are not prominently present in the past history. Alcohol, tobacco, and hard work cannot be important factors, since there is a higher percentage of female patients of this selected group than in the whole number of tabetic cases. In the cases giving a definite history of syphilis there had been insufficient treatment after infection.

SURGERY.

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Cure of Hydrocele by Extraserous Transposition of the Testicle, after Incision, Retroversion, and Retrofunicular Suture of the Tunica Vaginalis.—GENOUVILLE and PERAIRE(*Annales des maladies des organes genito-urinaires*, 1907, xxv, 481) in a report of 45 cases calls attention to 4 cases in which the injection of iodine for the radical cure had failed. The injection in the first had caused an hematocele with the loss of the testicle; in the second it was followed by a recurrence; in the third there were three vaginal concentric pouches with a failure of the iodine to extend to all, and thus a failure; and in the fourth there was a hernial sac filled with a translucent liquid, besides a small vaginal hydrocle. The iodine was dangerous to the hernial sac and did not reach the hydrocele. Of the many operations for hydrocele, Genouville and Peraire consider the two most important to be, (*a*) excision of the sac and (*b*) retroversion and fixation of the sac. They have employed local anesthesia with cocaine in all their cases. For anesthesia of the testicle and vaginal tunic a concentrated solution of antipyrine was applied, which completely suppressed the sensibility of the testicle. After making the scrotal incision into the tunica vaginalis the testicle was isolated from below upward with a portion of the cord. The two edges of the tunic were then turned backward until they met behind the cord, where they were sutured together, beginning at the lower pole of the testicle, turned upward, and continuing the suture to the upper pole of the testicle, where the edges of the tunic were made to surround the cord closely. Not infrequently after this operation the vaginal tunic has folded on itself to form a new vaginal cavity and a new hydrocele. To avoid this the writers make a subcutaneous pouch to the internal side of the scrotal wound, in which the testicle is placed and where it contracts adhesions to the surrounding cellular tissue. The wound is then closed and a strongly compressive dressing applied. They have had no recurrences.

Hermetic Suture of the Bladder, by Detachment and Turning in of the Mucosa.—DELBET (*Annales des maladies des organes genito-urinaires*, 1907, xxv, 518) says that while an open wound of the bladder tends to close spontaneously, it does so slowly in many cases, and that the general tendency now is to close it primarily when there is no special contraindication. He is not inclined to accept the reports of some surgeons that primitive and total reunion is the ordinary result of their operations. More frequently he believes a partial closure by cicatrization and a limited fistula is the result, with total closure later. In many cases the effort is a complete failure. The alteration from tension to flaccidity due to the filling and emptying of the bladder causes the edges of the wound at the time of tension to be slightly separated and urine to leak through. This happens in spite of the retained catheter, which is frequently blocked by clots of blood. A wound of the peritoneal surface closes as easily as a similar wound of the intestine. In default of the serous surface in extraperitoneal wounds, the mucosa is rich in bloodvessels and produces rapid cicatrization. Delbet closes a hypogastric extraperitoneal wound of the bladder as follows: The edges of the wound in the mucosa are caught with forceps, and with a bistoury or scissors the mucosa is separated from the muscularis around the wound for about 1 cm. With interrupted sutures or a continuous suture the muscularis is caught on both sides at the limits of the denudation, and at the time of tying them the edges of the mucosa are turned inward and their raw surfaces approximated. The internal hydrostatic pressure forces the raw surfaces closely together, rendering them water-tight. This method was employed in 2 cases with primary union in both.

A Contribution to the Study of Spontaneous Cure of Tuberculosis of the Kidney.—Deschamps (*Annales des maladies des organes genito-urinaires*, 1907, xxv, 589) says that for some years the surgical therapy of renal tuberculosis has been the order of the day, and that the majority of surgeons consider that early radical operation, nephrectomy, is the only operation capable of producing a cure. Dubot, for instance, said that, potentially, renal tuberculosis may be spontaneously cured, but that no case has been demonstrated anatomically and that clinically it does not exist. Rafin said that the pseudocures are examples of either a total destruction of the kidney or an obliteration of the ureter which excludes the kidney from the rest of the organism. Deschamps says that renal tuberculosis, like tuberculosis elsewhere, can be cured spontaneously, and that to propose a nephrectomy at once when tubercle bacilli are found in the urine, is the same as to advise an amputation of the thigh for a white swelling of the knee. Besides, early removal of one kidney does not prevent tubercularization of the other. The indications for nephrectomy given by the writer are as follows: When the whole kidney is invaded by the tuberculous process and there is little or no parenchyma left; when there is a coincident existence of a pyonephrosis and signs of general infection; when hematuria is abundant and repeated; when the painful crises are frequent and when there is a progressive cachexia caused by tuberculosis of the kidney. He reports two cases of cure, in one of which a nephrectomy was done with drainage, and in the second case medical treatment and a trip to the country were followed by a complete cure.

A Case of Early Intervention for Severe Burn of the Stomach.—Lambotte (*Jour. de chir. et annales de la Société Belge de chir.*, 1917, vii, 175) says that the ingestion of a caustic liquid produces, in a considerable proportion of cases, isolated lesions of the stomach wall, without involving the mouth or œsophagus. It may prove severe enough to be fatal. When the patient lives, two kinds of results may ensue: those occurring in the days immediately following the accident, and the later results from cicatricial stenosis, which may appear some weeks or years after the burn. In Quenu and Petit's analysis of 94 cases, 59 with autopsy reports and 34 with operation, there was not a single case showing early operation, that is, in the first days after the operation.

March 1 last a young servant swallowed a considerable quantity of what is thought to have been commercial hydrochloric acid. Very severe pain in the abdomen and vomiting followed immediately and on the following day hematemesis. Ice was applied to the abdomen and the pain calmed by morphine. In the succeeding days vomiting became incessant, the pain continued to be acute, and the feeding was impossible. March 5, when Lambotte first saw the patient, the condition was very grave. The temperature was 38.6° C.; vomiting incessant, of fetid and brownish material; pulse rapid; abdomen very painful, tender, and rigid. There were no signs of burn of the mouth or œsophagus, but the lips were slightly ulcerated. Immediate operation was advised to remedy a menacing perforation and to permit feeding, and was performed an hour later. The pyloric half of the stomach was injected and thickened, and gave the sensation of a tumor on palpation. This tumor ceased abruptly at the pylorus and diminished insensibly toward the middle of the stomach. No trace of perforation was seen. Posterior gastro-enterostomy was performed, with a very large anastomotic opening. The gastric mucosa was absolutely necrosed, and detached itself in fetid and black shreds. On the following day the patient's condition was satisfactory. Diuresis was reëstablished and vomiting had ceased, and the general condition fairly good. After four days the patient became distinctly convalescent, and on the nineteenth day the cure was complete. Lambotte is convinced that without operation a fatal termination would have ensued.

Complete Rupture of the Left Kidney; Partial Nephrectomy; Cure.—Lambotte (*Jour. de chir. et annales de la Société Belge de chir.*, 1907, vii, 177) reports a case in which a boy, fifteen years old, fell his length on the pavement, striking his left kidney region on the edge of a stone. He had severe pain which rapidly subsided. It returned, however, and with it came an attack of mild syncope. He passed a small quantity of urine, with some blood in it. Rupture of the kidney was diagnosticated and he was taken to the hospital. When Lambotte saw him, six or seven hours after the accident, he found distinct rigidity, but a good general condition. An incision in the loin showed a large quantity of clotted and liquid blood. The kidney was rapidly delivered through the wound and found to be badly lacerated. The upper third of the organ was removed. The remaining lacerated portion was repaired by catgut sutures and ligatures, and the wound drained with guaze. The wound oozed abundantly at first, and later suppurated; but the final result was very satisfactory.

The Treatment of Joint Ankylosis by the Transplantation of Plates of Cartilage.—Weglowski (*Zentralbl. f. Chir.*, 1907, xxxiv, 481) says that, unfortunately, we are far from obtaining full function by our treatment of joint ankylosis, and that usually we purposely aim to obtain an ankylosis in good position. Attempts to procure a movable joint have been made by placing between the joint surfaces foreign tissues, such as fasciæ, fatty tissue, muscle, or various foreign bodies like celluloid, gold plate, etc. Weglowski has employed plates of cartilage for this purpose on a case of elbow-joint ankylosis, upon which he had already operated unsuccessfully. The joint surfaces adhered together soon, in spite of the systematic employment of gymnastic exercises. He then concluded to try the transplantation of cartilage. After separating from the joint structures for a considerable area the soft tissues and periosteum, he sawed through the mass, at about the lower epiphysis of the humerus, with a Gigli saw set in an ordinary saw-frame. Then the upper end of the ulna was cut out, the head of the radius shelled out, and the remaining excess of bone was removed. He then exposed the cartilages of the sixth and seventh ribs, and removed two plates of cartilage together with the perichondrium in the whole length and breadth, but only half the thickness of the cartilages. These two pieces were then placed between the newly formed bony surfaces, the perichondrium being turned toward the epiphysis of the humerus. They did not require special means of attachment, since they were held in place by the adjacent bony surfaces. The wound was closed by suture throughout and a plaster cast was applied. After ten days the dressing and sutures were removed, showing healing by first intention. From this time on active and passive movements were carried out. In a month 60 to 70 degrees of flexion and extension could be performed easily, while pronation and extension were completely normal.

Unfortunately the patient died, five weeks after the operation, of pneumonia. This, however, permitted an examination of the joint operated on. The transplanted cartilage was found fixed firmly to the humerus. The apposed bony surfaces were smooth, even, and slightly glistening. Microscopic examinations showed new blood-vessels between the perichondrium and the bone. The cells and their nuclei of the cartilage stained with various stains.

Veronal-chloroform Narcosis.—Poktilo (*Zentralbl. f. Chir.*, 1907, xxxiv, 505) recommended this combination for general anesthesia in an article published two years ago. Since that time it has been employed in Professor Diakanow's clinic in Moscow, and has given entirely satisfactory results. The dangers of chloroform and ether may be met by decreasing the quantity or by using an antidote, especially for the evil effects of chloroform on the heart. This combination has the additional advantage of overcoming the effect of excitement of a nervous patient from the prospect of an approaching operation. It likewise lessens the quantity of chloroform necessary and diminishes the after-effects, such as headache, nausea, and vomiting. Veronal produces sleep without any evil effects, and causes the appearance in the urine of "amido" (NH_2), which, like ammonia, acts favorably on the heart. Given 1½ to 2 hours before the operation, it will put the patient into a sound sleep or a partial sleep by the time he is brought into the operating room, so that he will be indifferent to the operation. A comparison was made between the effects of three combinations with chloroform, bromethyl-chloroform, hedonal-chloroform, and veronal-chloroform. The patient was anesthetized, on the average, with the first in 5.21 minutes, with the second in 3.82 minutes, and with the third (veronal) in 2.58 minutes. Excitement was observed in the first in 45.08 per cent. of the cases, in the second in 27.17 per cent., and in the third (veronal) in 22.70 per cent. The average duration of the excitement was 2.89 min. in the first, 1.87 min. in the second, and 1.30 min. in the third (veronal). Vomiting was observed in 16.83 per cent. in the first, 6.79 per cent. in the second, and in 2.30 per cent. in the third (veronal). Asphyxia occurred in 7.62 per cent. in the first, 4.53 per cent. in the second, and 1.15 per cent. in the third (veronal). In one minute on the average there was consumed, in the first 0.69 c.c., in the second 0.57 c.c., and in the third (veronal) 0.61 c.c. The relatively better results from the veronal-chloroform combination deserves that it be further employed and its effect carefully noted.

The Technique of Intestinal Suture.—Schoemaker (*Zentralbl. f. Chir.*, 1907, xxxiv, 508) considers, from his experiments on dogs, that the end-to-end union of divided intestine with circular suture gives better results than the end-to-side or the side-to-side or lateral anastomosis, and that it should, therefore, be preferred in men. Intestinal resection is the more dangerous the more complicated the suture employed. The chief difficulty met with is in closing properly the interval between the two layers of peritoneum of the mesentery where it is attached to the bowel. Schoemaker's method is as follows: These two layers of peritoneum slightly separated near the gut, corresponding to each divided end of the intestine, are brought together and turned in as when the intestinal end is to be closed, by two sutures. On one side the threads are cut close to the knot, and on the other side are left long, without regard to the rest of the divided mesentery. One long end of the suture nearest to the bowel is then employed as a circular suture to unite the two ends of the intestine. This suture is made to catch the serous, muscular, and submucous coats or all the coats, and when it completes the circumference of the bowel it is knotted to the other long end of the same suture and cut short. The second suture, that farthest away from the bowel, is similarly used as a circular suture, including only the serous coat, and thus covering in the first suture. Schoemaker employed this method in 10 cases without a failure. It is not so successful in uniting the divided ends of the large intestine, since the non-peritoneal surface of this bowel is larger and the edges of peritoneum too far apart.

Stab Wounds of the Heart.—Harte (*Annals of Surgery*, 1907, xlv, 672) says that the consensus of opinion among experimenters is that the heart after being exposed can be grasped with the hands or forceps and gently compressed with no appreciable effect on its action; that punctures with needle or knife produce only a temporary irregularity in the heart's action; that wounds produced during systole bleed more than those occurring during diastole; that wounds of the ventricle produced during systole are larger than those produced during diastole; that oblique wounds bleed more than perpendicular wounds; that wounds of the right ventricle are more dangerous, because of the thin ventricular wall and because blood in the right heart coagulates more slowly; that wounds of the heart heal kindly, and that the cicatrix is complete in two weeks; that interrupted sutures are better than continuous ones; that the material enclosed in the grasp of the sutures causes atrophy and is replaced by scar tissue; that superficial stitches are less likely to tear out than deeper ones, and that the stitches should be inserted and tied during diastole, because of the danger of tearing out during systole.

Fisher, after an analysis of 452 heart wounds, says that from 7 to 10 per cent. recover spontaneously. Harte thinks this estimate high, but considers that even if it were positive it should not deter one from prompt surgical interference if the condition of the patient warrants it. He believes that an anesthetic is imperative and that ether is preferable. The best suture material is chromicized catgut, reasonably fine, introduced on a sharply curved needle. In ventricular wounds the suture should be inserted deeply, even to entering the endocardium, as only by this method can accurate approximation be procured. In auricular wounds through-and-through sutures are imperative to prevent bleeding through the wound. The pericardium should be closed without drainage, the pleura with drainage in a dependent part of the chest. Harte reports a case in which a stab wound of the left auricle was sutured. The patient died twenty-four days after the operation, from infection of the pleural cavity, which was opened by the original stab wound.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Duration of Immunity following the Prophylactic use of Diphtheria Antitoxin. STILLER (*Jahrb. f. Kinderheilk.*, 1907, lxiv, 142) from a year's observations at the Strasburg Children's Clinic concludes that prophylactic immunization lasts from three to five weeks provided the subjects are removed from infectious surroundings. If immunized children are subjected to possible infection, the immunity is naturally of shorter duration—ten to fourteen days for example. Children living in these surroundings are far more prone to contract the disease than those to whom antitoxin has been administered. Catarrhal affections and traumatisms of the mucous membranes predispose to infection and shorten the duration of immunity. Scarlatiniform eruptions which appear without fever or throat lesions, Stiller considers to be the manifestation of true scarlatina, a conclusion which hardly seems justifiable.

Radiotherapy in Leukemia.—VAQUEZ (*Revue française de médecine et de chir.*, 1907, v, 4) states that radiotherapy is our best means of combating the course of leukemia. It is without effect in the acute forms of the affection, but in the subacute and chronic types it may hold the disease in arrest. Radiotherapy is to be considered as a symptomatic measure only and has given no proofs that it is a cure; its injudicious employment, on the other hand, may be followed by grave results to the patient. It should be administered only up to the point of disappearance of the pathological modifications of the blood, to the exclusion of leukopenia. Every relapse should, upon its appearance, receive the same treatment as was given the disease originally. Methodical examination of the blood should be carried out in making the diagnosis, in following the course of the treatment during the active period of the disease, and in anticipating the occurrence of relapses.

Digitalis and Digitalin.—HUCHARD (*Revue de thérap.*, lxxiii, 24, 838) protests against the use of substitutes for crystallized digitalin. He states that it is not true that the infusion of digitalis leaves is preferable to the glucoside from a point of view of diuresis, for the latter possesses this effect to the same degree as the former. Digitalis varies greatly, depending upon its origin, the soil in which it grows, the method of the preservation of the leaves, etc.; all these render any accurate test of its action impossible. Consequently, a product of uniform strength is always preferable. The slowness of the drug's action can hardly be considered a fault; diuresis follows its administration in from twelve to forty-eight hours while cardiac stimulation takes place in fifteen minutes; these two effects are absolutely unconnected. It must be remembered that digitalin is diuretic only when œdema is present. The drug is not dangerous when properly employed, its action is lasting and its administration may be continued for months. 5 minims of a 1 to 1000 solution may be given daily as a cardiac tonic for long periods. Its elimination is retarded when the patient is kept in bed. The dose varies with the mode of employment and the indications; 50 minims of a 1 to 1000 solution during the day have a diuretic effect and may be repeated after eight or ten days if necessary. If such a dose is followed by a disappearance of the œdema the drug may be continued as a heart tonic in the doses indicated above. Digitalin may be given hypodermically in oily solution if there is likelihood of causing gastric disturbance.

Practical Value of the Opsonic Index in the Treatment of Disease.—CRACE-CALVERT (*The Lancet*, 1907, 1, 279) states that in ordinary sanatorium patients suffering from tuberculous disease of subacute type and whose progress is not what we should reasonably expect, inoculations of tuberculin, controlled by the opsonic index, may do a considerable amount of good. In such patients there is probably an absence of stimulus to the immunizing machinery and there is accordingly no corresponding production of antitoxins. If, now, by inoculating, the necessary stimulus is given a corresponding production of antitoxins, accompanied by a rise of the opsonic index and an improvement in the lung condition, should be evidenced. The index, however, does not always respond, for the author in one instance has observed a fair amount of improvement in the pulmonary condition without much effect on the opsonic index. The appropriate doses of vaccine for a given patient can be learned only by experiment and repeated estimations of the index, but it has been proved that the doses ($\frac{1}{500}$ of a milligram and upward) recommended in the directions given with Koch's tuberculin are too large and better results can be obtained with smaller dosage. The dose generally employed by the author is $\frac{1}{1000}$ of a milligram. The frequency of the inoculations depends upon the length of time that the positive phase, following inoculations, lasts. At first the injections are usually given at seven to ten-day intervals, while, as the number of inoculations increases, the frequency is diminished until a positive phase lasting two months after an inoculation of $\frac{1}{800}$ of a milligram, may be noted.

The Effect of Amyl Nitrite Inhalations upon the Blood Pressure.—HEWLETT (*Jour. Med. Research*, 1907, xv, 383) after experimentation states that amyl nitrite increases the total output of blood from the heart both during the primary fall of systolic pressure and during the secondary rise. During the former the increased out-put is due to the more rapid heart action, during the latter mainly to a greater amount of blood expelled at each systole. It would consequently appear that though the drug dilates the peripheral vessels it does not necessarily reduce the blood pressure to a like degree. The latter is avoided by an increased output of blood from the heart. The typical action of amyl nitrite is not that usually ascribed to it, viz., dilatation of the peripheral arteries and fall of blood pressure. It does, indeed, dilate the bloodvessels, but it also causes the heart to pump more blood and the result is a more rapid flow of blood through the body. With regard to variations from the typical action of the drug the author has observed that the pulse rate in old persons, whose hearts are comparatively free from vagus control, does not become as accelerated after an amyl nitrite inhalation as it does in the young. In discussing the action of glyceryl nitrate Hewlett has observed, in patients who received a daily dosage of from 5 to 30 drops of a 1 per cent. solution, that following such dosage only a slight and inconstant effect on the blood pressure occurs. No great fall resulted, indeed, the systolic pressure rose about as frequently as it fell. When this drug causes the pulse to become "fuller and more bounding" Hewlett is inclined to believe that we are dealing with an effect similar to that of amyl nitrite. The action of glyceryl nitrate in relieving certain headaches and in reducing the number of attacks of angina pectoris is not simple, and it is possible that its effects in these conditions are not due to a resulting lessening of blood pressure but to an increased circulation of the blood.

Methylthionine Hydrochloride in Mercurial Stomatitis.—LAMOUREUX (*La Semaine médicale*, 1907, xxvi, 586) has used this agent in mercurial stomatitis as follows: Once a day, preferably in the morning, he applies powdered methylthionine hydrochloride to the ulcerations of the mucous membrane by means of a cotton swab. The mouth is then rinsed with a 2 per cent. solution of potassium chlorate; the latter procedure is repeated several times during the day. Of 6 patients thus treated 4 were cured by the fourth day, while the other 2 recovered within eight days. It is interesting to note that, in instances in which mercurial treatment must be energetically carried out, it need not be intermitted if this treatment of the stomatitis is employed.

The Treatment of Whooping-cough.—Blache (*Journal de médecine de Paris*, 1906, xviii, 512) recommends the following: Syrup of tolu, syrup of senega, syrup of eucalyptus, equal parts; dose 4 to 8 drams daily. During the convulsive stage the patient is given small frequent meals, after the third of which one or two spoonsful of black, sweetened coffee, which is held to be an excellent anti-emetic, are taken. During the catarrhal stage ipecac may be prescribed; this drug is most efficacious when the bronchitis is but slight.

Belladonna gives variant results. Blache employs it thus for patients less than one year old: Belladonna extract $\frac{1}{6}$ grain, powdered belladonna root 4 grains, sugar 8 grains. Mix and divide into twenty-five powders. From one to four of these should be given daily, the number being increased progressively. Children of from two to five years may receive six to eight powders per day. Good results may also be obtained by a combination of belladonna with potassium, sodium, or ammonium bromide in syrup. Drosera will also diminish the number and intensity of the paroxysms when given as follows: Tincture of drosera 40 parts, tincture of aconite 10 parts, tincture of belladonna 8 parts. Dose 20 to 50 minims daily, divided into 3 doses. Change of climate and air-baths are excellent means of hastening recovery.

The Prevention or Scarlatinal Nephritis.—Thompson (*Edinburgh Med. Jour.*, 1907, xxi, 103) states in regard to the use of a prophylactic drug against the nephritis of scarlatina that urotropin is the only one which has proved itself of value. Of 47 consecutive patients treated with this agent by the author not one had nephritis and only one exhibited albuminuria. These facts he considers encouraging enough to suggest the further use of the drug in scarlatina. Patients treated with hexamethylenetetramine were less fortunate in escaping nephritis, and he holds this drug to be not an exact equivalent of urotropin when considered from the point of therapeutic results. In conclusion, he suggests that all instances of scarlatina be treated from the beginning with 5 grain doses of urotropin three times a day for children up to twelve years; for patients above this age $7\frac{1}{2}$ grains thrice daily are advised. Generous dilution of the drug with water is necessary and the treatment should be continued to the end of the twenty-eighth day of the disease.

The Trypsin Treatment of Cancer.—Luther (*New York Medical Journal*, 1907, lxxxv, 341) states that the consensus of opinion of all those who have applied the treatment is that whatever else it may do, it certainly does, in most cases, cause: (1) An arrest or shrinkage of the growth; (2) improvement in the general nutrition in which the appetite is increased and the weight is maintained or augmented; (3) diminution or cessation of pain; (4) diminution in discharge with a decrease of fœtor; except in patients in whom sloughing occurs. In conclusion Luther emphasizes the fact that large doses (75 minims of a sterilized glycerin extract of freshly macerated pancreatic gland) are required in order to obtain the best effects. The agent is administered hypodermically, and the injections may be given anywhere except into the tumor. They should be thrown deep into the cellular tissue but not into the muscular substance. Thorough asepsis should be observed and the syringe should be cooled after being boiled, for heat precipitates the trypsin.

The Treatment of Subacute and Chronic Gout.—LUFF (*Practitioner*, 1907, lxxviii, 161) states that in the subacute and chronic forms of gout, in addition to colchicum, which may be given in small doses, guaiacum is often useful as an alterative; here it stimulates the liver and affords relief to the portal system. It is given in the form of the resin in doses of 5 to 10 grains, two or three times a day, according to the effect on the bowels. In chronic gout the colchicine pill may be given three times daily. To encourage elimination through the kidneys, diuretics, such as potassium citrate or bicarbonate should be prescribed and water should be freely drunk. The citrate is the best potassium salt to combine with colchicum. It diminishes the acidity of the urine and increases the solvent power of this fluid for the uric acid salts. Mineral waters containing sodium salts are useful if the liver is sluggish, and, when there is gastro-intestinal catarrh, torpor or gouty dyspepsia. The lithium salts are less useful than those of potassium or sodium. The former may cause cardiac depression or even dilatation if they are taken in excessive amount. In order to lessen the articular thickenings and enlargements, the gouty pain in the side of the foot and gouty neuralgia, the internal administration of potassium iodide is advised. Its dosage should be 10 grains three times a day for six to eight weeks, and it is best given in a decoction of sarsaparilla.

PEDIATRICS.

UNDER THE CHARGE OF

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A Urinary Sign of Helminthiasis.—IEFIMOV (*Bull. méd.*, abstracted in *Revue des maladies de l'enfance*, January, 1907, p. 45) describes a urinary reaction which indicates, without fail, he states, the presence of intestinal worms: 5 to 10 c.c. of urine is received directly in a test tube, and without filtering (at least if not markedly turbid) is heated to boiling, and then 5 to 10 drops of the officinal solution of acid nitrate of mercury is added. In negative cases the urine becomes cloudy, lactescent, and throws down a white precipitate. In cases of helminthiasis this turbidity, as well as the precipitate, always takes on a grayish or even blackish tinge, due presumably to the presence of a toxin elaborated by the parasite, which is absorbed by the blood and finally eliminated by the urine. To ensure the accuracy of the test it is indispensable that the subject shall not have recently taken certain drugs, especially alkalies, compounds of sulphur (including Dover's powder, which contains the sulphate of potash), certain iron preparations, and lead compounds, all of which give with the acid nitrate of mercury a grayish or blackish precipitate. This is not the case, however, with morphine, the salicylates, carbolic acid, salts of zinc and copper, phenacetin, quinine, and antipyrin. Purulent urine alters the reaction, but the presence of albumin, sugar, or indican does not interfere with it. This reaction is said to be produced by the presence of teniæ as well as of round worms. The grayish coloration of the precipitate is always more marked in the case of the tape-worm, but the difference is not sufficiently distinct to indicate the variety of the parasite. The differential diagnosis, however, can be made, according to Iéfimov, by a microscopic examination of the crystals obtained by evaporation of a thin film of the urine upon a glass slide. In the case of tape-worms there are a number of granular crystals; in the presence of nematodes, star-shaped crystals are found, which are particularly large and beautiful when ascarides are present in the bowel.

OBSTETRICS.

UNDER THE CHARGE OF

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The Results of Active Interference in the Treatment of Parturient and Puerperal Cases.—Hofmeier (*Monats. f. Geb. u. Gyn.*, 1906, xxiv, Heft 4) discusses this important subject at length. 3.3 per cent. of the children are stillborn. Of these, 1.9 per cent. perish during labor. These cases may be divided into two classes: First, those where the bony pelvis occasioned no complication in labor; and second, those in which contraction of the pelvis, with or without other complications, brought about the result. In the first group are 56.6 per cent. and in the second, 43.3 per cent. of cases. During labor 16 per cent. of the children born in contracted pelves perished. This mortality has been decreased of late years by operations increasing the size of the pelvis and by Cesarean section. The infant mortality of induced labor is very unsatisfactory. Krönig estimates that among 440 labors, 2.5 per cent. terminated in fetal death. When interference was practiced in labor, fœtal mortality arose to 20 per cent., and during forceps extraction was 12 per cent. Hofmeier's figures show 10 and 2.5 per cent. respectively in the Würzburg clinic. Among eleven children delivered by pubiotomy and Cesarean section, 9 per cent. perished. In the Würzburg clinic, 66.6 per cent. of labors in contracted pelves, terminated spontaneously; 33.4 per cent. required interference of some sort. Among these patients 26 per cent. of the children perished, but this occurred in cases which were not treated by Cesarean section, or operations for enlarging the pelvis. Maternal mortality in cases in which the children perished during labor is usually very small. In 163 cases in which interference other than Cesarean section or pubiotomy was practised, no mother died. In 27 cases in which operation was undertaken in the interests of the children as well as the mother, 3 died—one after symphysiotomy, one after pubiotomy, one after Cesarean section.

Care of the Perineum and Pelvic Floor. APFELSTEDT (*Berl. klin. Woch.*, November 26, 1906) endeavors to prevent injury to the perineum during labor by placing the patient upon the back with the thighs widely separated. The left hand supports the perineum with four fingers, the forearm being supported upon the bed. Two fingers are placed upon each side of the coccyx. The fingers of the right hand are pressed between the occiput and the pubes, controlling the exit of the head. Unless the patient is controlled by anesthesia at the moment of birth, she is asked to count when strong expulsive pains come on, thus reducing the danger of laceration by strong voluntary expulsive efforts. Should a tear occur, a curved needle is passed deeply into the torn muscular tissue from the centre of the wound close to the margin. This is carried out on both sides, a heart-shaped suture being passed and buried.

Labor Obstructed by Osteosarcoma of the Pelvis.—RAY (*Brit. Med. Jour.*, January 26, 1907) reports the case of a patient who gave birth to a child eighteen months before coming under observation. During the present pregnancy she had suffered from pain in the hips and on examination was found to have lost weight and to be very costive. When labor developed, a tumor was found near the sacrum as large as a child's head. Under ether an incision was made and a tumor found, smooth, tense, and elastic. The tumor was opened and found to be not fluid, but solid. Its removal could not be effected. Two hours after operation the patient had bleeding, and labor gradually developed. This was terminated by the delivery of a dead child by version. The child was stillborn, and the mother also perished shortly afterward. On examination, the tumor was found to be an osteosarcoma of the sacrum and coccyx.

The Influence of Meat Diet upon Fertility and upon Lactation. WATSON (*Brit. Med. Jour.*, January 26, 1907) has experimented upon rats, some of which were fed upon meat and the others upon milk. Those fed upon meat remained sterile, while those fed upon milk conceived and were able to nourish their young.

Postpartum Hemorrhage. LE PAGE (*Brit. Med. Jour.*, January 26, 1907) has treated postpartum hemorrhage successfully by the following method: The patient's pelvis is raised considerably. The abdominal aorta is compressed against the bodies of the vertebræ; the legs and arms were held up and bandaged. The hand was made thoroughly aseptic, introduced within the uterus, and clots removed. Ergot was administered, and brandy and water injected into the rectum. The pressure upon the aorta was continued, and very gradually removed. Care was taken also to remove the bandages from the legs and arms very gradually.

Cesarean Section for Contracted Pelvis Complicated by Twin Pregnancy.—Cowen (*Brit. Med. Jour.*, January 26, 1907) reports the case of a primipara, aged thirty-five years, with a rachitic pelvis, whose true conjugate was about three and one-third inches. As it was impossible to deliver the children through the vagina, they were extracted by abdominal incision. At the request of the parents, to prevent further conception, the Fallopian tubes were also excised. There were two placentæ which occupied the greater portion of the uterine cavity. Mother and children recovered.

Scopolamine and Morphine to Produce Analgesia during Labor.—In the report of the proceedings of the Obstetrical Society of Berlin (*Zeitschr. f. Geburtshülfe und Gynäk.*, 1907, Band lix, Heft 1) Hocheisen quotes Viron and Morel as reporting 2000 cases of this anesthesia, with 25 deaths; Roith, 4000 cases with 18 deaths; Wartapetian, 50 per cent. of the children asphyxiated; and Gauss, in Krönig's clinic, 500 cases with good results. Hocheisen and Bardeleben report between 120 and 130 cases. The drug produced an apparently light sleep, from which the patient was aroused. Gauss had good results in 76 per cent. of his cases. Hocheisen and Bardeleben had negative results in 18 per cent., and the influence upon the patient produced by the drug did not seem to be in proportion to the dose. In 65 per cent. the desired light narcosis was obtained. Very decided after-effects, as redness about the head, thirst, and rapid pulse, were observed in 70 per cent. of cases. The physician must not leave the patient while under the influence of the drug, as unpleasant symptoms often develop. Uterine contractions were observed in a considerable number of cases. In 24 per cent. abdominal pressure was distinctly lessened. The application of forceps was necessary in 25 per cent. of cases. The length of labor was increased. Postpartum hemorrhage was more frequent, 1 case proving fatal, and the time occupied in the delivery of the placenta was longer than normal. The puerperal period was also complicated by subinvolution. After labor, 1 patient had severe disturbance of the heart and seemed to be in great danger of heart failure for several hours. In 20 per cent. of the cases there was slowness of the pulse and intermittent beat. The action of the heart and the respirations were disturbed in many of the children. One fatal case was observed, which seemed fairly to be ascribed to the drug given. In the discussion of this report, Gauss agreed in the dangers of the method, and Bumm thought that the method was not suitable for general practice.

What is the Exciting Cause of Labor?—This interesting question receives considerable attention by Schatz (*Archiv f. Gynäk.*, 1906, Band lxxx, Heft 3), who believes that the phenomena of menstruation and labor depend upon rhythmical variations, producing a maximum and minimum in blood pressure. The tendency is to divide the various periods of the mouth into halves. Especially is this true in the last three half-months of pregnancy, and in the month extending from January 19 to February 18, thence to the week of confinement, or even the half-week preceding. The physiological week is not a primary division of the physiological month. Periodic activity in the bodily functions seems to have a definite relation by division in multiples of two. The physiological week is one step in the subdivision of the physiological month. A short month would be twenty-one days between menstruation, usually divided into four periods. The more early and complete the division of the month's curve of blood pressure, the greater the liability in the regularity of the periods of the given patient. When these variations become excessively frequent, the intervals being minutely divided, it is considered a sign of what the author terms "angioneurasthenia." Lack of this excess of division is characteristic of a well-balanced vasomotor nervous system. The illustration of this theory is observed in a case of epilepsy. When conception occurs a new periodicity begins. An effort may be made to predict the time of confinement by studying the blood curve of the patient. Labor is most apt to take place during the period of increased blood pressure, and as this varies in different patients it is obvious that each case must be studied individually, to attempt a prediction.

The Secretion of Sweat from the Axillary Glands Resembling Milk, and Occurring during the Puerperal Stage.—SEITZ from the clinic at Munich (*Archiv f. Gynäk.*, 1906, Band lxxx, Heft 3) reports two cases of this unusual abnormality. From the second to the fourth day of the puerperal period these patients complained of pain in the axillary region, with sweating on both sides beneath the skin. The tissues seemed to be infiltrated, the veins engorged, and the whole axilla filled, but without redness. On pressure sweat exuded from the axillary region. Microscopically this fluid closely resembled milk. These cases were not polymastia in the axillary region. There were no nipples or mammary glands developed, and the fluid could definitely be recognized as sweat.

Placenta Circumvalata and Marginata.—LIEPMANN (*Archiv f. Gynäk.*, 1906, Band lxxx, Heft 3) considers this condition as caused by defective development in the membranes. Placenta marginata develops from placenta circumvalata. It is probable that a condition of endometritis precedes these abnormalities, and that the infarcts which form are secondary to defects in the membranes. These abnormalities are the result of an unusual location of the placenta, or some abnormality in the amniotic liquid, or in uterine contractions occurring during pregnancy. The condition clinically is not of importance as regards the third stage of labor.

GYNECOLOGY.

UNDER THE CHARGE OF

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Medical Treatment of Uterine Hemorrhage.—AARONS (*Brit. Gyn. Jour.*, 1907, No. 2), in a paper on this subject, presents a series of 93 cases in which various drugs were used before resorting to curettement.

He emphasizes the importance of ascertaining the cause of the bleeding before resorting to any treatment. He found a combination of ergotin, cannabina tannate, hydrastin, and stypticin especially useful. Styptol, $\frac{3}{4}$ to 2 grains three or four times daily, checked hemorrhage in all but two cases in which it was used. Aarons thinks that it neither produces uterine contractions nor does it act on the vasomotor system. Calcium chloride was particularly useful, by increasing coagulability of the blood, especially in cases of fibroid tumors. No styptic applications to the interior of the uterus were made except after curettement.

In several instances hemorrhage was checked by rectal injections of sterilized gelatin, given three times daily, according to Lancereaux's suggestion with regard to treating aneurysm by causing coagulation of the blood. Hot douches were rarely used.

Conservative Treatment of Inflammatory Diseases of the Adnexa.—Haerrmann (*Münch. med. Woch.*, 1907, p. 145) reports 1244 cases from the Munich Clinic, in only 20 of which were the organs extirpated. He infers that operations may be necessary in cases of acute inflammations of the tubes and ovaries and pelvic connective tissue in which life is endangered. In all cases of tuberculosis a radical operation should be performed; also when conservative treatment has been tried without benefit. In only 7 out of 180 cases was such surgical treatment necessary. Although gonorrhea was inferred to be the cause of disease in the majority of the cases, the pus evacuated was nearly always sterile; gonococci were seldom present, streptococci more frequently. Intestinal infection was thought to be the cause of the adnexal disease in several instances. Haerrmann lays considerable stress on the blood count as an aid to the diagnosis of suppurative processes. The conservative treatment included the pressure and hot-air methods; massage was rarely used and electricity never. Absolute rest in bed, hot douches, and tampons were employed as routine measures. The average stay of the patients in the hospital varied in different kinds of cases from 37.3 to 55.7 days. In only 36 cases was vaginal section necessary in order to evacuate pus.

Intestinal Obstruction due to Parametritis.—Kuliga (*Monat. f. Geb. u. Gyn.*, vol. xxiv, 1906, p. 598) reports 3 cases in which exudates caused almost complete occlusion of the rectum and subsequently intestinal paralysis. He cautions against treatment by heat and pressure, which cause fresh inflammation, and also points out the danger of perforating the wall of the gut with the long rectal tube. He advises that an artificial anus should be made instead of operating directly for the obstruction.

Fusion of an Ovarian Cystoma with the Uterus.—Meyer (*Zentralbl. f. Gyn.*, 1906, No. 44, p. 1239) reports a case in which the uterine wall was partially transformed into a multilocular cyst, which seemed to originate in the uterus itself. The well-defined line of separation between the uterine muscle and the neoplasm and the fact that the exterior of the cyst was covered in several spots with serous epithelium showed that it was really of ovarian origin and had invaded the uterus secondarily. The lining membrane of the cystoma consisted of columnar epithelium secreting mucus, which resembled closely that of the intestine and thus suggested that the neoplasm was doubtless a teratoma of the ovary.

Results of Vaginal Extirpation of the Cancerous Uterus.—BLAU (*Beiträge zur Geb. u. Gyn.*, Band x, Heft 3) presents the statistics of Chrobak's clinic from 1890 to 1893. Of the cases of cancer of the uterus 20.46 per cent. were regarded as operable, 18.5 per cent. being suitable for vaginal hysterectomy. There were 309 cases of vaginal extirpation (87.7 per cent. for cancer of the cervix and 11.65 per cent. for malignant disease of the corpus uteri), the primary mortality being only 5.8 per cent; 244 patients have been kept under observation, 161 of whom were regarded as promising a favorable result. Of the latter 30.4 per cent. are still free from recurrence. The proportion of absolute cures has been estimated by Winter as only 5.64 per cent.

There were 113 cases of igni-extirpation (in 47 of which Schuchardt's incision was employed), with a mortality of 6.19 per cent., the proportion of those permanently cured being 45.8 per cent. (Such remarkable results as these obtained by so reliable an observer as Chrobak must impress American gynecologists not only with mixed envy and admiration, but with a mournful sense of the small number of operable cases which are submitted to them as compared with the proportion in German clinics. This unfortunate condition is due directly to the fact that neither the profession nor the laity in this country has yet learned to recognize the early signs of malignant disease of the uterus.—H. C. C.)

Papillary Tumors of the Ovary.—ESTOR (*Gaz. des hôp.; Zentralbl. f. Gyn.*, 1906, No. 44, p. 1242) admits that the etiology of these neoplasms is unknown. He distinguishes a benign and a malignant variety, the latter developing in younger subjects, but both between the ages of forty and forty-five. The former may remain benign for a long time, then may suddenly undergo malignant degeneration and form metastases.

Both ovaries are nearly always affected and ascites is a common accompaniment, while adhesions, metastases, disturbances of the intestinal tract and of menstruation are usually present. Papillary cystomas may exist for several years before the patient succumbs, and the prognosis is favorable even after an incomplete operation (?). As regards operative treatment Estor recommends total extirpation of the uterus with the cysts if evidences of malignancy are noted; otherwise, supravaginal amputation. When ascites exists drainage should be maintained for three or four days. The primary mortality is high—in Estor's cases 15 per cent. (It would seem as if he failed to distinguish clearly between benign and malignant neoplasms, especially with regard to the indications for radical operation. It is important clinically to note that there is a wide difference between true metastases and papillary growths found in the peritoneum in cases of non-malignant cystomas.—H. C. C.)

Infection of Ovarian Cyst with Eberth's Bacillus.—BENSIS (*Orient. méd.; Zentralbl. f. Gyn.*, 1906, No. 44, p. 1243) reports the case of a patient, aged forty years, who had had an ovarian cyst for sixteen months which was mistaken for pregnancy. Two and one-half months before entering the hospital she had typhoid fever, during the course of which the tumor rapidly increased in size. Purulent fluid removed from it with an aspirator contained Eberth's bacilli. The temperature was normal. The patient's condition was such that a radical operation was impossible. Twelve pounds of pus was removed with a trocar, but the patient died a few hours later.

Cysts of the Tubal Mucosa.—CORNIL (*Bull. et mém. de la Soc. anat. de Paris; Zentralbl. f. Gyn.*, 1906, No. 44, p. 1244) removed a cystic ovary with a tube the size of the thumb. The ovary was a unilocular cyst with papillary growths on its inner wall, covered with columnar epithelium. The mucous membrane of the tube contained many small submucous cysts, which appeared to be deep depressions of the mucosa, covered with cylindrical epithelium and surrounded by connective tissue. Cornil thinks that this condition must be rare, as it has not been previously described.

Intraligamentary Cysts.—SJANOSZEWSKI (*St. Petersburg Dissert.; Abst. in Zentralbl. f. Gyn.*, 1906, No. 44, p. 1244), from an analysis of nearly 500 cases, concludes that it is impossible to define exactly the point of origin of these neoplasms, whether the ovary, parovarium, or broad ligament. They usually develop between the ages of twenty-five and thirty-five years, and in 36 per cent. in nulliparæ.

The general primary mortality after operation is 10.9 per cent., due to frequent adhesions, injury to the hollow viscera, and the escape of infected cyst fluids. Subsequent exudations are common and result in a mortality of 2.5 per cent.

Colpotomy is the preferable operation, being easier, attended with less risk of traumatism than laparotomy, a shorter period of convalescence, and fewer subsequent complications. The vaginal route is especially convenient for the removal of suppurating cysts, as the peritoneal cavity is not invaded; partial removal of the sac with drainage is best accomplished per vaginam.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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Intranasal Origin of Epilepsy.—HEYNINX, of Brussels (*Archives internat. de laryng., d'otol., et de rhin.*, March–April, 1906), reports four cases in support of his contention that inflammatory reaction at the vault of the nose and in the subcranial sinuses may be the cause of epileptic seizures in consequence of the retention of septic secretions due to hypertrophic and congestive compression of the subjacent tissues, and especially the middle turbinate.

Nasal Disease a Main Cause of Facial Erysipelas.—CULLEN F. WELTY (*Jour. Amer. Med. Assoc.*, December 22, 1906) contends that so-called idiopathic erysipelas is a misnomer. He states that 82 per cent. of cases of facial erysipelas start from the angle of the nose and are due to direct infection from a contaminated field of pure or mixed infections of streptococci, which can be substantiated in the majority of instances. These views were confirmed in a discussion.

Transformation of Nasal Mucous Polyps into a Melanotic Tumor.—GEORGES PAUTET (*Revue hebd. de laryng., d'otol., et de rhin.*, December 1, 1906) reports a case in which he was enabled to follow the slow evolution of transformation of a mucous polyp of the nose into adenoma and finally into melanosarcoma. This occurred in a man fifty-two years of age, who first came under observation September 13, 1900, and, after a number of surgical procedures at varying intervals, eventually died in cachexia on May 10, 1904.

Operations on a Denuded Nasal Septum.—E. ESCAT (*Revue hebd. de laryng., d'otol., et de rhin.*, November 3, 1906) reports and illustrates one case in which he gained access to the septum by cutaneous incision between the lip and the point of the nose, followed by denudation of the cartilage and its removal with simple scissors. The cutaneous incision was united with sutures which were removed on the fourth day. Three months later both the physiological and esthetic conditions were excellent.

Turbinectomy.—E. HARRISON GRIFFIN (*Med. Record*, April 14, 1906) states that he has operated so far on more than ten thousand cases without having seen one in which the result was not most gratifying, and the aim of the operation secured—perfect nasal breathing and a drain for the nose and all the sinuses that empty into the nostril. He removes only enough of the bone to establish drainage and to avoid the possibility of contact with the septum, finding it unnecessary to remove the entire hypertrophied bone. Before operating, he administers quinine and potassium bromide for one to two weeks to reduce congestion and diminish risks in hemorrhage. In operating he uses a saw, with the aid of forceps, snare, and scissors, utilizing cocaine anesthesia, but avoiding adrenalin.

Pemphigus of the Mouth and Pharynx.—M. HECHT (*Revue hebd. de laryng., d'otol., et de rhin.*, October 20, 1906) reports the case of a man, aged forty-four years, who for six months had an affection of the buccal and pharyngeal mucous membranes, manifested by the almost daily appearance of multiple yellow vesicles of the average size of a pea, which ruptured rapidly, leaving painful losses of substance. It extended at times to the epiglottis and to the arytenoid region, and had resisted all medication up to the time of presentation. On account of the absence of fever at each eruption, and the lack of manifestation in the skin, objection was taken to the diagnosis in the discussion which followed.

Adenocarcinoma of the Turbinates.—FIOCRE (*Revue hebd. de laryng., d'otol., et de rhin.*, October 20, 1906) reports a case of adenocarcinoma of the nose in a woman, aged sixty-three years, who complained simply of dulness of hearing, and was unaware that her right nasal fossa was occluded with a growth. This was removed, principally in one sitting, and a month later the passage seemed entirely clear, cicatrization being nearly complete, and nothing discoverable but healthy tissue covered with normal mucous membrane. The tumor occupied the position of the middle and inferior turbinates without invasion of any of the adjoining tissues, and had begun to undergo myxomatous degeneration.

Diseases of the Accessory Sinuses in Ophthalmic Affections.—HASTINGS (*Annals of Otol., Rhin., and Laryng.*, September 1906) discusses the subject, and reports a number of illustrative cases in point. He enumerates six ocular conditions other than orbital abscesses, as those most frequently met with in this connection, namely: Œdema of the eyelids, congestion of the conjunctiva, ptosis, squint, pain in the eye, and disturbance of vision.

Insufflation of Oxygen Gas under Pressure in the Treatment of Empyema and Sinusitis of the Maxillary Antrum.—ORESCO (*Ann. des maladies de l'oreille, du larynx, du nez, et du pharynx*, November, 1906) reports eleven successive cases successfully treated by intranasal puncture, washing with bichloride or phenol solution, and followed by insufflations of compressed oxygen gas under two hundred and fifty pounds' pressure.

Clinical Forms of Cancer of the Maxillary Sinus.—SEBILEAU (*Ann. des maladies de l'oreille, du larynx, du nez, et du pharynx*, November, 1906), based upon some thirty cases observed by him in the course of four years, of which but two were surviving, divides the clinical forms into three: neoplastic, suppurative and infectious, and putrid or gangrenous. He shows that the suppurative form is often mistaken for maxillary sinusitis or for pansinusitis, and that the mistaken diagnosis is sometimes inevitable.

The Tonsil as a Focus for Tuberculosis.—CHAS. M ROBERTSON (*Jour. Amer. Med. Assoc.*, November 24, 1906) gives an anatomical description of the tonsil and reports his researches, tracing direct infection from the tonsils through the cervical lymphatics to the lymphatics of the pleura and thence to the apex of the lung. In studying 232 patients whom he had operated upon, he found that 8 per cent. presented undoubted tuberculous lesions in the form of giant cells and tuberculous-tissue change, the greater number of tonsils having been of the so-called submerged type. He therefore advocates the complete extraction of the diseased tonsils. This he does either under cocaine anesthesia or ethyl chloride preferably, with specially devised scissors, of which illustrations and descriptions are given.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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The Cause of the Increased Portal Pressure in Portal Cirrhosis.—HERRICK (*Jour. Exp. Med.*, 1907, ix, 93) has investigated this problem by injecting washed livers, and has observed the pressure influences of the hepatic and the portal circulations upon one another in normal and cirrhotic livers. The arterial and portal injections were varied together as to their pressures, then the arterial circulation alone, and finally the portal circulation alone, were studied in order to ascertain their behavior under various pressure conditions. It was found that the portal circulation was as free in cirrhotic livers as in normal livers, thus casting doubt upon the generally accepted ideas of the cause of ascites in cirrhosis. Herrick found that when the pressure in the hepatic artery was raised from 0 to 130 mm. Hg. the pressure in the portal vein in normal livers rose but 3 or 4 mm. Hg., while in cirrhotic livers this rise of pressure in the hepatic artery produced a rise in the portal vein of 20 or 30 mm. Hg. This is due to the fact that in cirrhotic livers there is a far greater communication between the arterial and portal currents than in normal livers.

Herrick believes that the factors contributing to the increased portal pressure in portal cirrhosis are: (1) The direct communication of the arterial pressure to the portal vessels through dilated capillaries, and (2) the larger volume flow of the hepatic artery in proportion to the portal flow in cirrhosis as compared to that in the normal liver. When salt solution was injected into the hepatic artery it was found to return by the portal as well as by the hepatic vein, while from a portal inflow the return was through the hepatic vein only. The portal pressure has a decided influence on the volume flow in the hepatic artery, and *vice versa*. This influence was found to be more marked in cirrhotic than in normal livers. The communication of the arterial pressure to the portal pressure is an important factor in explaining the increased portal pressure in portal cirrhosis.

The Pathology of the Liver in Cardiac Disease, and its Clinical Lessons.—SALAMAN (*Lancet*, 1907, i, 4) discusses the changes that take place in the liver when it has been subjected to stasis of blood and back flow. He advances the idea that the liver acts physiologically as a safety-valve to the heart, and that this action is definitely altered by the changes produced by congestion. He finds that a normal adult liver will take up fluid equal in volume to that of the liver itself, if the portal vein has been tied and the injection is made through the hepatic vein, at a pressure of two feet and three inches of water, the pressure of the right ventricle. When a pressure of one foot of water, that equal to the pressure of the right auricle, is maintained, the liver takes up fluid equal in volume to about one-half or three-fourths of its own volume. When the pressure is released the liver recoils by its own elasticity and about two-thirds of the injected fluid is expelled. Salaman believes that with the aid of the intra-abdominal pressure it is possible for all of the absorbed fluid to be expelled.

This experiment was then tried on three types of livers which had been subjected to engorgement for longer or shorter periods. With only recent engorgement and with fatty but no fibrous changes, it was found that the capacity of the liver for taking up injected fluid was not impaired, while the recoil was much less than normal. In livers moderately fibrosed, congested and nutmeg in type, the capacity for absorbing fluid was found distinctly reduced, while there was an increase in the recoil of the liver when the pressure was removed. When the cirrhotic nutmeg liver, with portal, hepatic, and central fibrosis, was used it was found that its capacity for absorption was still more reduced, and that the recoil was not so great as in the second type.

From these facts Salaman concludes that under conditions of cardiac stress the liver will draw off a large volume of blood from the right auricle, and that after a time this property of distensibility is distinctly impaired by fibrous changes, which changes, however, increase the force of the liver's recoil and prevent its use as a reservoir for blood. In complete cardiac compensation there is no engorgement of the heart and no back pressure. While the liver may be looked upon as a sponge-like safety-valve to the heart, continued use of this safety-valve action leads to its own abolition. Salaman believes that liver pain, œdema, and ascites in heart disease may be symptoms of broken compensation in the liver.

The Leukolytic Action of the Blood Serum of Cases of Leukemia Treated with X-rays, and the Injection of Human Leukolytic Serum in a Case of Leukemia.—Capps and Smith (*Jour. Exp. Med.*, 1907, ix, 51) have carried on a series of experiments to determine the effect of the *x*-rays upon the leukocytes and the serum in cases of leukemia. They first injected normal human serum into rabbits and guinea-pigs, and found that this caused a rise of leukocytes in twenty-four hours of from 5 to 15 per cent. A second series of experiments showed that when the serum of cases of lymphatic leukemia untreated by *x*-rays was injected into rabbits and guinea-pigs, a greater increase in the leukocytes occurred than with normal serum, their increase being from 5 to 56 per cent. When serum from cases of leukemia treated with *x*-rays was used, however, a marked decrease in the number of leukocytes in the blood of rabbits and guinea-pigs was found. In a case of the lymphatic type, in which improvement in the clinical picture of the disease was greatest, the reduction was from 35 per cent. to 60 per cent., while in a case less improved the reduction was only from 10 to 20 per cent. In a much improved case of splenomyelogenous leukemia the leukocytes were reduced 12 per cent. and again 40 per cent.

Capps and Smith conclude that the amount of leukolytic substance present probably varies with the amount of improvement observed clinically in the cases. They exposed defibrinated human blood, both from healthy individuals and from cases of leukemia *in vitro* to the *x*-rays and found practically no diminution in phagocytic power of the leukocytes. They think it improbable, therefore, that Quadrone is right in saying that several exposures of animals to the *x*-rays increase their resistance to infection by stimulating phagocytosis.

The effect of leukemic serum on human leukocytes and erythrocytes was studied by mixing these cells with the serum in hanging drops. It was found that leukemic serum which had not been exposed to the *x*-rays had no leukolytic action, nor were the erythrocytes changed by it. The serum from *x*-rayed leukemic cases, however, was found to have a marked leukolytic action on leukocytes from normal individuals and from cases of leukemia. In the latter case the action seemed selective, and showed a marked preference for mononuclear cells and myelocytes. This serum agglutinated the red blood cells in the hanging-drop preparations. The *x*-rays were found to destroy leukocytes and produce a leukolytic substance in leukemic blood *in vitro*, but no such reaction was obtained with normal blood. The number of leukocytes in the circulating blood of leukemic cases was found to decrease after exposure to the *x*-rays. In order to test the action of the leukolytic serum on an untreated case of lymphatic leukemia the serum from a treated case which was known to be highly leukolytic was injected into the abdominal wall of an untreated case. Three injections were made, and each time there was a distinct reduction in the number of leukocytes by destruction principally of the mononuclear elements, but the reduction was always followed in a few days by a rise to or above the number before the injection. Capps and Smith think this leukolytic substance is probably formed by the destruction of the leukocytes.

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ORIGINAL ARTICLES.

THE SIGNS OF PRE-DEMENTIA PRÆCOX: THEIR SIGNIFICANCE AND PEDAGOGIC PROPHYLAXIS.[1]

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There is an ancient tradition that in oriental lands it is the duty of the practitioner of medicine to keep his patients from falling ill, and that the rewards that accrue to the physician in the Celestial Kingdom are in proportion to the number of days of health enjoyed by his patron. Further, the family doctor is held responsible, pecuniarily as well as morally, for the time lost in illness. While we recognize the fictitious character of this procedure, nevertheless, were it so, it would represent an ideal that is fortunately coming to its own in occidental lands—that prevention is the great goal toward which the science of medicine has turned its chief attention.

That birth and death cannot be modified will not be questioned so long as the race endures, but in the brief life span between, may not much of the sorrow and distress of sickness be removed by enlightened prophylaxis? Even the casual observer knows how fruitful the theme has been already, and the enthusiastic optimist believes he can see the passing of much preventible sickness in the near future. That which enlightened hygiene has done for the limitation and repression of the infectious diseases; what careful dietetics is doing for the control of disorders of metabolism; what orthopedics and gymnastics are doing for the relief and cure of physical defects, may not adequate psychological pedagogy do for certain forms of mental disorder? Rational mental teaching has shown its value in hysteria, and in many neurasthenoid and psychasthenic states. Is there a possible application of its principles for the very early stages of that which today we term dementia præcox, and, if so, what are the sources to which we must look for help?

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[1] Submitted for publication, January, 1907.

The problem is far from being a new one in any sense of the word. It is as old as there are records of human observations, and in the past few centuries Willis,[2] Rousseau, Morel, Maudsley, Falret, Down, and others have been insistent on calling attention to the phenomenon, and each in turn attempted to awaken an active interest leading to the solution of a medicopedagogic problem propounded of old. At the present time there is little let-up in the question, but, for us at least, the leading minds are approaching it from the pedagogic rather than from the medical side, so much so that it almost seems that Hall's severe criticism is partly justified, at least when he writes[3] that "the years of puberty have been too much neglected by the physician. The years of senescence and of maturity have a rich literature, but there is a relative neglect of the morbid peculiarities of pubic life." And this is the most important period of human life, for at this time the sunlight of nature bursts its bounds and radiates into all the human faculties, stimulating them to their adult form. This is the period left too much to itself, trustfully and hopefully; sometimes, it must be confessed, neglectfully; or, as Meyer has well suggested,[4] "fatalistic has often been the attitude of those who have accepted hereditary tendencies as excuses for the bad conduct of children, and they have been left uncared for by reason of a lack of faith in those who should exert themselves the most to save them from themselves."

Meyer further writes: "Do not let us say that this (he is speaking of dementia præcox) is merely one of nature's methods of weeding out the unfit. Among the 25,000 persons who are today (1903) in the public and private institutions of New York State alone, there are many brilliant hopes buried, largely owing to a lack of knowledge of what some people need in the way of social and personal hygiene. Remember that some of the most illustrious members of the race have been dangerously near the borderland of insanity, and seem to have been great, although they showed obvious traces of the same misled instincts that completely wrecked others. Are such people not worth our help? Should not the home, the press, and the school mind some of the dangers and shape their ethics and methods accordingly?" That they are doing so is evident to anyone who will but glance at the current literature of school hygiene, wherein we see reflected the great effort that is being expended for a solution of many of the problems suggested.

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[2] Willis, 1672. "Many young people, in childhood intelligent to a degree and extremely teachable, so that by their learning and conversation they swept everyone into a state of admiration, afterward as they grew older turned out stupid and dull, and those who before were very beautiful in person afterward presented no grace or comeliness in their appearance." See also Morel, under head of syndrome called by him "Stupidity."

[3] Adolescence.

[4] Child Study Address, 1903.

Of these mental affections, that of dementia præcox offers some hope of progress. Just what is here meant by the term dementia præcox I shall not attempt to define further than by saying that the word stands for a provisional group of patients who gradually develop certain mental and physical characteristics first brought into special prominence by Hecker and Kahlbaum, amplified by Pick, and later developed and expanded by Kraepelin and his students—a group very clear as to its nuclear features, but misty in its outlying edges and far from constituting an unassailable synthesis—a fact well recognized by Kraepelin himself. Still, by its erection clinical psychiatry has been a great gainer, and the whole problem of types of personality has had a new light thrown upon it, which promises much for all investigators. The question at issue, however, is not what constitutes a diagnosis in mental disease;[5] but the fact that there are in some individuals certain deteriorating processes at work which result in fairly definite symptom-complexes or pictures is the feature that needs emphasizing.

Granting for the time being the possibility of a fairly constant and characteristic type of degeneration leading to the various grades of mental enfeeblement, the problem narrows down then to a consideration of the question, Are we in a position to make not a diagnosis of dementia præcox in the usual sense—for then the process has been too long in the making—but are we able in the light of newer investigations[6] of the types of individual psychological reactions to the surroundings to say that such and such a child is liable to undergo this particular type of mental stagnation or deterioration, and is it possible by taking proper heed of the little things to avoid a wasted life, or conserve in some degree, sufficient for ordinary needs, an individual who otherwise would pass to utter destruction?

To the asylum physician who with wide-open eyes works with the dead end-results, the dements, the question at first sight seems hopeless; to the teacher who with eyes half-opened sees the early signs of unnatural tire, unreasoning irritation, overreaction to slight stimuli, the inquiry is large with anxious hope.

Most observers who have studied dementia præcox have been struck by the relationship of the symptom picture at the outset as well as in the advanced cases to the normal features of adolescent development. No one has expressed it better than Hall when he writes: "There are two missing links indispensable to a full acquaintance with the many forms of precocious mental decay; first and chiefly, knowledge of the actual changes during the stage of puberty and later adolescence, which go on within the limits of sanity. This age has often been characterized as that of mental and moral inebriation and of psychic madness, and from my studies of its phenomena I am convinced that nearly all of its symptoms can be paralleled in the inner and outer life of youth who do not lapse toward the terminal imbecility, but develop to sane and efficient maturity. Here we may demand that the normal psychologist shall help to contribute a sound basis to the study of disease, while the alienist, who deals only with salient, flagrant, or ostensive instances where natural tendencies are 'writ large,' and who does not know the genetic and natural history of the soul, cannot solve the most fascinating problems presented by clinical pictures."

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[5] As to the validity of diagnosis in mental disease, A. Meyer (Brit. Med. Jour., 1906, p. 757), contributes a discussion of more than usual value: "Diagnosis is merely a convenience of nomenclature, an expression for a group of facts. In psychiatry the facts occur in very complex combinations, and therefore a one-word diagnosis is almost sure to fall short of what it ought to do, namely, short of presenting the actual facts in the case, sufficiently to designate the etiologic, prognostic, and therapeutic status. Psychiatry has not yet reached and probably never will reach the stage where a small number of one word diagnoses would be more than a formal index," etc.

[6] Illustrative cases are here omitted for sake of brevity. See Masselon's excellent study, Thèse de Paris, 1902, and also Jung's more recent monograph issued after this paper was prepared, Ueber die Psychologie der Dementia Præcox, Halle, 1907.

Whether we can follow Hall to the full limit of his suggestions is doubtful; nevertheless the thought that he has so well amplified from the writings of others and so well summarized is deserving of the most careful attention.

The suggestions of Bleuler, Meyer, and others, that in these trying years of change many fall by the wayside for lack of a proper adjustment to the reactions of everyday life, offers a most fruitful key to the answer of our problem. The work of Hall and his students, of Marro, of Spitzner, of Ludwig v. Strümpell, and of Heller, not to mention a long list of previous and contemporary workers, has served to bring into prominence the altered states of mind in this restless period of development, and in the fruitful phrase of Meyer, "habit psychosis," there is more than a mere suggestion.

Puberty is the time of sudden expansion of personality and individuality. The boy or girl commences "to feel his oats," and parental counsel, especially if unwisely urged, is scorned and spurned. The individuality commences to assert itself, and in our modern times, and among urban youths, ideas of fortune and renown commence to fill the mind. Coe has called attention to the acute exacerbation of the feeling of self-consciousness of this period as well. He shows that reflectiveness often leads to self-criticism that may become morbid. "Anxiety to do right exhausts the energy that should go to action, trifles are augmented to mountains, or debate with one's self as to what is right is carried so far as to paralyze decision." This increased introspection and heightened period of self-consciousness is invariably attended by ideas of being under observation, and furtiveness and oversuspiciousness is a trait that may become fixed unless corrected in a wise manner. To draw within one's self is with some the very opposite tendency to those bolder self-assertive souls, yet with both, dreams morbid and moody, or castle-building, are characteristic of the period. Thoughts of overesteem, overconfidence in one's ability, here may lay the foundations in the adolescent for ideas of superiority and even insane conceits.

Imagination may know no bounds. At puberty this inner mental fire seems to be kindled into the most exuberant and least restrained forms. Illusions are many and varied, and often their making becomes so habitual a pleasure-giving device as to resist subsequent uprootal when the youth is brought face to face with life as it exists for everyday purposes. This is the time when all of the senses feel the touch of poetic flight, and sound and color pictures fill the minds of many. Hall writes that now "thought and the two higher senses (sight and hearing) are never so vivid and intense, for they are just ready to bifurcate, each to lead its own life, so that the two are now peculiarly liable to be confused, intellectual processes to take sense forms, and vice versa. It is for this reason that hallucinations, false sight, and especially hearing, are most common; but in brains tainted by morbidity the faculty of discriminating mental from sense forms is undeveloped, and dreamy conditions may become habitual, so that we see here the natural budding of insane perceptions."

Consecutive speech in the form of conversation, particularly with strangers, is at this time being demanded of the adolescent. New school associates commence to widen the horizon, and when we would analyze a conglomerate of many individuals we perceive spreading out from the mean, comparatively small numbers, the divergent tendencies, either of excessive speech tendencies or reticence, both of which if uncorrected by the tendency of habit become stereotyped, and if the element of intellectual deterioration be added very significant symptoms, known to psychiatrists, stand out in bold relief. In many individuals at puberty we find a very marked tendency to catch at phrases which sound well, and the youthful mind in its imitative manner grasps expressions from many sources and packs them in the memory for the appropriate time to come, when they may be used to the best advantage. All are acquainted with the youthful types which use such grand, undigested phrases, sometimes to great advantage. They are often thought positively brilliant by admiring parents. With increasing mental power these exuberant growths become more moderate, or the general rise in the whole intellectual level makes what at one time were islands of extravagant thought and expression a part of the picture of more mature power, coördinated and regulated to the needs of speech, spoken or written. The psychiatrist is also only too aware of the significance of this symptom when it persists, either by reason—which one cannot decide on a priori grounds—of a loss of that rise in general mental power, or because of a marked "neural trend" on an exaggerated habitual exercise accompanied by a "reaction hypertrophy," if I may so characterize it, of this character. These patients show the peculiar affected speech, filled out with large-sounding phrases and words clearly out of their normal perspective (neologisms.) The more complex state of Forel's "word salad" defies analysis from this simple standpoint.

Inasmuch as youth has not gathered enough facts of life on which to base wide judgments, it is very natural that at this period, when some reaction is necessary, we should find a sudden blossoming out of the imitative faculty. This in the motor sphere shows itself very distinctly in mannerisms, modes of walking, etc. One can almost picture the youths of a definite college group by their swing and their swagger; their imitations, further, conscious or unconscious, of modes of dress, of brushing the hair, choice of neckties, etc., etc. In a small number one sees these reactions become stereotyped—as it has been well expressed, "just as speech may lose its highest function and lapse to not only slang, but even interjectional forms, so poses may slowly become fixed postures and even contractures, or a limited repertory of stereotyped acts may develop on a basis of the natural automatisms of Lindley."[7] Whether one is justified in reading into those individuals at the other extreme speech negativism as an exaggerated habit reaction, independent of the processes of deterioration, or of reaction to delusional thoughts, is still an open question; yet the general process of negativism so characteristic not only in adaptation of the muscles of speech, but in all motor reactions, will undoubtedly have much light thrown upon it if closer observations can be made of the pre-dementia peculiarities of these queer personalities with which we are all familiar.

That this question is not incapable of analysis Bleuler's[8] recent contribution to the subject of negativism will amply indicate. He discusses the simple mechanisms of the body, showing that throughout opposing forces are at work. In the muscles agonist and antagonist oppose one another and determine correct functioning. In the nervous system stimulant and depressant mechanisms modify the exchange of heat, the movements of the intestines, the dilatation and the contraction of the bloodvessels, and the sphincteric controls. Everything in life is conditioned by a play of opposing forces, of opposing ideas, of opposing effects. Conflicting emotions are aroused simultaneously, and there seems to be a definite mechanism at work. The child who hangs back on being offered a piece of candy illustrates this play of forces, and the control of the sexual impulse in prudery by rejection is almost universal. Vacillation is the rule, and the mechanisms are useful ones to prevent disaster from undue hasty action, quite comparable to the "synaptic phase" of the reflex arc which is conservative of too rapid passive extension of muscles. Many individuals in spite of marked intelligence cannot call up any opposing ideas because of the force of the impulses pushing to action, while others are unable to decide among the many contradictory impulses; they are light-headed and hypervacillating because of the the lack of definite impulses. The crying in joy and the laughter in sorrow, so commonly seen in hysterical subjects, for example, are illustrations of this loss of capacity of regulation.

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[7] Some Mental Automatisms, Ped. Sem., July, 1897, p. 41–60

[8] Die negativ Suggestibilitat, ein physiologisches Prototyp der Negativismus, der contraren Autosuggesstion, und gewisses Zwangsideen, Psy.-neur. Woch., 1904, Nr. 27, p. 249.

When the mechanism of opposition becomes too pronounced, becomes quantitatively stronger in function, negativism results. In physiological conditions one sees the contrary idea enter into active appearance as a form of negativism or as a suspicion often in combination with a distinct weakness of reflection. Children, many women, old women, show this form of physiological negativism. From the standpoint of teleology one can regard the early appearance of the contrary idea as a form of protection against being taken by surprise. The one who never permits its influence in daily affairs is our friend, Mr. Easy Mark. His contrast is seen in the confirmed pessimist, also a newspaper acquaintance, this time "from Missouri." Suggestibility and negativism, then, are to be regarded as different sides of a fundamental quality of the psyche which spring from a common source.

Jung and Ricklin have shown, in their studies on dementia præcox, that many of the most striking symptoms are only qualitatively or quantitatively distorted mechanisms of the normal soul life. This is very characteristically true of this symptom of negativism, of which there is unquestionably more than one kind. In dementia præcox, as I understand Bleuler, the primary idea, which may give rise to its normal opposing idea, is present, as in the normal mechanism; the play of different motives (deliberation) being retarded, only the primary and its contrasting impulse remain. Interference or retardation occurring in the primary impulse, an abnormal activation of the contrary impulse results, deliberation becomes impossible, and negativism, impulsive ideas, or obsessions prevail. There exists, then, not only a positive suggestion, that is, a tendency to accept the called-up ideas and feelings from the outside, but also an analogous negative suggestion, that is, a tendency to decline. This is normally one of the most important moments for the forcing of a deliberation to action. On the pathological side it leads to negativism, to contrary autosuggestion, and to a definite class of compulsory ideas.

The distractibility of youth is marked; especially is this marked in certain individuals. It is difficult for the child to keep its attention centred on one line of thought or action. With keen sense perceptions the child is open-eyed and open-eared to all the world. Not all children, however, by any manner of means. The impulses that determine a definite and adequate interest are often lacking in early life, and develop only with the onset of puberty and adolescence into any formal system. Here again the variation in child life in the matter of attention will repay careful analysis. Our statistics regarding the reaction types of attention in those who have gone to an early decay are not yet gathered, and definite criteria are lacking. That marked attention disorders are significant in dementia præcox is a commonplace. Distractibility is very common at times, although rarely exhibiting itself in the same manner as with the manic-depressive. The inability to concentrate on any one point, whether as significant of extraordinary fatigability or as indicative of the perpetuation of an adolescent reaction, cannot be as yet postulated. In view of the marked lack of interest that influences the process of attention, it seems not improbable that some other approach to an understanding of this attitude is needed.[9]

Looking back over a series of years and reviewing the early histories of a comparatively large number of patients whom I have known fairly intimately, both in their home life, their school, their college, work, etc., my own observation has led me to register a mental note that most of the precocious dements with the paranoid tinge have occurred in those whom I have known to be singularly lacking in the divertibility of the attention, particularly for objects of auditory and optic sense. These young people I have known have all been very poorly gifted in the power of external observation. They, as young men and young women, never saw anything in the ordinary sense of seeing. Their lights were all turned within, and while all were singularly gifted their gifts were made manifest in matters of literary or artistic constructive qualities, using the products of introspection rather than in the direction of their being natural science workers. This observation is in need of careful scrutiny, and is of the broadest character and is recorded simply as such.

The story of the emotional life of the adolescent is an unwritten book, so many are the intricate shades in various individuals; but in all youth there is a markedly increased emotional tone given to all the spheres of activity. With increasing powers of self-control the emotional reactions become less violent for the many, but here it is an observation of old, that intellectual training has little influence on the emotional life of an individual. They seem to be lower arc reflexes. The subtle series of reactions, whatever they be, that constitute the emotions are in a most peculiar sense individualistic, and one of the most primary qualities of personality.

The variabilities in the matter of the emotional life have not been overlooked. Studies from the anthropological side—of primitive man, even of animals—as in Darwin's well-known study of the emotions in man and the lower animals; Hughes, Mimik, De Sanctis, and others show that in this general psychological category we must look for the greatest amount of variability. Child study has emphasized how widely the children in a single family may vary in this as in other important aspects; hence it is not at all surprising to find one boy of seventeen often a comparatively unemotional personality from lack of an excitable emotional mechanism, while another with a much more variable instrument has learned to control, and still a third goes into adult life before control is gained, and an "hysterical tendency" finally eliminated by wise counsel, or by "experience" with the world. The various difficulties attendant on the study of our emotional nature, which Hall[10] infers is unfortunately too much taken up in the study of books by our regular psychologists, is widely appreciated.

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[9] Compare Madeleine Pelletier, L'association des ideés dans la manie aigue et dans la debilité mentale, Paris, 1903

Emotional stability undoubtedly can be acquired by proper training, and it is one of the great services of Dubois, of Bern, that he has formulated some of the fundamental notions whereby control of an unstable emotional organism can be acquired.

What Kraepelin considers as a fundamental sign of dementia præcox, "emotional dementia;" that which Stransky[11] has laid so much stress upon, namely, the great disproportion between emotional reaction and ideation, his dissociation of Thymopsyche and Noopsyche, what has been termed intrapsychic ataxia; or what Meyer has expressed[12] as "discrepancies between the mood and the reaction," is of special interest in this comparison of the not unusual reaction type of the mentally healthy and the mentally ill adolescent. In its relation to the emotional states of many hysterical personalities are offered some deep-seated analogies that should not be overlooked. If I might express it crudely, in dementia præcox we find a stage of emotional impoverishment due to the breaking down, perhaps as Freud terms it, a process of conversion,[13] of a rich plexus of associations of many years' growth—the changes in the affective life should be interpreted from the standpoint of a disintegration or analysis of what had heretofore been a developing and fairly fixed personality. On the other hand, in the emotional status of the hysterical I believe we have the raw material of emotional reaction unchecked and poor in associative control, not from a breaking down of connections previously made, but from a deficiency, or retardation, in development of these. To characterize the former as Kraepelin does, as an "emotional dementia" suggests the term "emotional backwardness," as a prototype of the other, and just as in organic nature very closely related products may be formed, the one by analytic or destructive processes, others by synthetic or constructive stages, so in the emotional sphere the symptoms of many individuals suffering from dementia præcox come to resemble those of patients with hysteria. That very difficult point of doubt where it is impossible to determine whether one has to deal with a simple cessation or a mild retrogression of function may some day be solved through a study of the adolescent emotional life and a study of the hysterical personality. The hysterical side of the problem is being approached by Breur and Freud from a study of the subconscious, by Ricklin and Jung in their association investigations, by Janet and Babinski and many others, and it seems not unlikely that, by a comparison of the broken-down personalities of the dementia præcox with the unknit and unstable personalities of the hysterical, light of lasting value will come into the problem. Just as Kraepelin came to such a heuristic idea of the general conception of dementia præcox by a study of the end stages—that great mass of secondary dements that seemed to contain so little hope of revealing any important principles of mental processes—so will that infinitely more hopeful study of personality in the formative periods of life afford measures by which the conception now gained may be amplified and clarified.

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[10] Adolescence, vol. ii, p. 59.

[11] Zur Kentniss gewisser erworbener Blodsinnformen. Jahrb. f. Psyc. u. Neur. 24, 1903, p. 1.

[12] Fundamental Conception of Dementia Præcox, Brit. Med. Jour., September 29, 1906.

[13] Ueber die psychischen Mechanismus hysterischer Phänomene, Neur. Ctblt., 1893, Nos. 1, 2, and later contributions.

In this hasty review the attempt has been made to bring to mind those normal adolescent features which by their "exaggerated development" or their "persistence" into adult life serve so to modify the personality of the individual as to give it an abnormal stamp—a stamp which has its alliances to many of the features which are recognized and grouped as dementia præcox, particularly as seen in hebephrenic and paranoid states.

In thus calling attention to these resemblances the important causes for the "exaggerated development" or for the "persistence" of these adolescent traits should not be lost sight of, and if the picture is to be rounded out in this respect one must not neglect to make an inquiry as to any antecedent factors which might have an influence, although it must be confessed that what is known is very meager and far from satisfactory.

Darcarren[14] has used the phrase "a characteristic prenatal tire" as being applicable to the precocious dement, summarizing, as it were, two at least of the most important subjects of inquiry, hereditary and fatigue influences.

Heredity is a vague term, and is used by us all in a very loose manner, notwithstanding the many pitfalls that have been pointed out by Weismann and the studies of special interest of Brown-Séquard, of Luciani, and of Fere.[15] Yet it is one of the most striking in the present connection, for among the psychoses dementia præcox shows abnormal antecedents in a disproportionately large number of instances, even exceeding the proportions of manic-depressive insanity according to some observers. Magnan speaks of the accumulation of hereditary influences in the ancestry[16] and makes a general series of categories for the "constitutional psychoses," as the French class them. Indeed, most modern French writers speak much of degeneration and heredity, but offer general remarks rather than actual facts. His paranoias are always (96 per cent.) tainted. Torrini's[17] phrase is that the psychoneuroses of the ascendants prepare the soil of the paranoid constitution of the descendants, and the term of his transition is his secondary paranoia. Tanzi and Riva[18] lay much stress on atavistic tendencies, thus still further introducing confusion into what is already very dark. One needs but to read the chapter of Anglade on the influences of heredity in Ballet's recent *Traité de pathologie mentale* to see how far from being coördinated are the recent French works at least. Gauthier[19] is one of the few of the French writers since Morel to give accurate data in this respect. He lays much stress on the hereditary factor in the causation of juvenile breakdown, and gives the histories of some 13 patients, all suffering from some one of the varieties of dementia præcox. Of these practically all show most marked hereditary ancestry; furthermore, in his cases at least 75 per cent. showed marked physical stigmas, usually multiple in character, cranial malformations, variations in eye-muscle balance (strabismus) being particularly common. In addition it was noteworthy that the patients for the most part suffered neuropathic accidents while young, particularly convulsions of a recurrent type. Indeed, the occurrence of motor anomalies is very characteristic in these patients, and is often foreshadowed while very young.

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[14] Thèse de Paris, 1902.

[15] Comptes-rend. Soc. de biol., March, 1882. Arch. Ital. d. mal Nervosi, 1881. Soc. de biol., 1894 et seq.

[16] Congres internat. de méd., 1890.

Ancient psychiaters were so impressed by this truth and made it such a commonplace that careful statistical studies have been neglected. Esquirol spoke of an adolescent insanity as characteristic in hereditarily implicated parents; Morel also spoke of the outbreak of insanity at the adolescent period as the revelation of the degenerate taint.

German students have contributed widely to the subject of the influence of heredity in dementia præcox. Kraepelin is among those who lay much stress on this factor. He says that at least 70 per cent. of the patients with dementia præcox show a definite hereditary taint, and in at least 17 to 18 per cent. the immediate ancestry, paternal or maternal, is involved. Illberg in his monograph finds, on the one hand, a much lower percentage, 45 per cent. of near and remote ancestry influences, in 20 cases subjected to searching analysis, while, on the other hand, in a higher percentage than Kraepelin's, 30 per cent., the influence was direct. Among English students Clouston[20] is most emphatic when he says: "The insanity of puberty is always a strongly hereditary insanity; it, in fact, never occurs except where there is a family tendency toward mental defect or toward some other of the neuroses." Clouston also calls attention to what he terms unphysiological methods of education as an important factor in the causing of these early insanities. "There is no time nor place of organic repentance provided by nature for some of the sins of the schoolmaster."

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[17] La paranoia secondaria, Revista Sperimentale di Freniatria 1889, vol. xiii, p. 52.

[18] Riv. Sper. di Fren., 1886 vol. xii.

[19] De la démence precoce chez les jeunes aliénés héréditaries, Thèse de Paris, 1883.

[20] Sixth edition, p. 582.

Numerous other studies might be quoted on this point, but the general concensus seems to show that heredity is an element of great importance in the breakdown of these adolescents.

What kind of hereditary influences are most to be feared, or, rather, are certain taints in an ancestry more likely to lead to dementia præcox rather than to other forms of mental disease? We are not yet in a position to state this with definiteness.

Mendel repeats in his text-book what has often been said, that similar types of insanity are more apt to be transmitted to the children, and in the light of the present time groupings, numerous studies tend to accentuate this general viewpoint. Thus, Strohmayer says[21] with a certain degree of positiveness, that it is characteristic that the same type of psychoses appears in family after family—the severe hysterias, paranoia, hebephrenia, periodic and circular psychoses, and imbecillity.

Krauss,[22] working with the material of the Heidelberg Clinic, states that he has found as high as 93 per cent. of similar heredity in dementia præcox in ascendants and descendants, but his figures are drawn from but comparatively few instances, 13 cases of dementia præcox in the ascendants showing 14 descendants with dementia præcox and 1 with manic-depressive insanity. In a group of 7 ascendants with manic-depressive insanity 11 descendants were affected with the same type of disease, 4 with dementia præcox. Krauss' table IV, showing dementia præcox in more than one member of the family, is particularly instructive.

Kalmus[23] points out that, in his studies of the Lübeck Institute, in the majority of cases of dementia præcox hereditary factors on both sides were present.

Pickett's[24] analysis of 58 cases of well-marked dementia præcox is of interest. He divides them into the classical groups: 30 of hebephrenic, 11 of katatonic, and 17 paranoid forms. In his 30 hebephrenics, direct mental heredity, including alcoholism, is shown in 17, over 50 per cent. Insanity occurred in the parents of 7, nervous disease (not stated) in 3, apoplexy in 2, and alcoholism in 5. Tuberculosis and pneumonia are listed, but the connection is too remote for our present purposes. In his 11 katatonics direct heredity is present in 6, insanity in father or mother being present in 3 and in 1 sister. In his paranoids the proportion is 10 in 17, direct insanity in the parents showing in 4. His figures are quite comparable to those of Kraepelin and others, although not supporting very strongly (indeed such a small number would hardly admit of searching comparisons) the view that the paranoid types show the highest percentage of hereditary taint. The type of insanity in the parents is unfortunately not given. In fact, such figures are difficult to obtain, notwithstanding their value and the comparative uselessness of those that are here given. Apart from Toninni's quoted statement that the psychoneuroses of the ascendants tend to appear as paranoia in the descendants, we are lacking light in this direction.

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[21] Allg. Zeitschr. f. Psychiatrie, 1904, 61, p. 365.

[22] Ibid., 1903, 60, p. 224.

[23] Ibid., 1905, 62, p. 230.

[24] Journal of Nervous and Mental Disease, 1904.

Vorster[25] has made specific inquiries in regard to both dementia præcox and manic-depressive insanity, and has presented an able summary of the work of his predecessors, to which reference may be made for the best survey of recent German literature.

Vorster has also made an attempt at a more definite classification of the insanities under discussion, and has given us perhaps the most satisfactory piece of work for our present purposes. He controverts the idea so wide-spread in previous times that the insanities of hereditary transmission are polymorphic in type, and arranges himself with those who see a likeness in the form of mental disease of the parent and the child. Only the recent groupings of Kraepelin could render his conclusions so valuable. He presents the histories of 23 families, in 9 with manic-depressive disease, in 8 dementia præcox, in 6 a psychosis of later life. In his 9 manic-depressive ascendants he finds 80 per cent. manic-depressives in the descendants, while in none of his families with manic-depressive ascendants were either dementia præcox or paranoid states found in the descendants. (Vorster has found 36 per cent. heredity of dementia præcox in 1000 cases.)

While most authors who have studied the question of heredity in mental diseases have stated that manic-depressive insanity shows the highest percentage of hereditary influences (Morel,[26] Kraepelin,[27] Sioli,[28] Fitscher,[29] etc.) it should not be forgotten that the precocious dement rarely has the opportunity to become an ancestor. On this account the problem in this disease acquires special interest, and special light is desirable to prejudge the influence of this factor. Vorster's study shows that all of the families with dementia præcox in the ascendants showed dementia præcox in the descendants, and that moreover in his cases there were more instances of inbreeding (the marriage of cousins) resulting in many more cases of dementia præcox than of manic-depressive insanity. Certain of Vorster's findings are of particular interest, showing that while the same general disease group was transmitted, it may appear in a different form. Thus in his family 1, in addition to the katatonic symptoms which were present in the twenty-nine-year-old mother and two sons, there was also a marked paranoid state in one of the sons. In family 2, a paranoid state was characteristic in the twenty-four-year-old mother (a cousin), while simple hebephrenoid dementia was present in both daughters. In family 3 mother and son showed marked impulsive stereotypy which in the mother was manifest in her "haltung," while in the son it appeared in his movements. In family 4 the excitement stages in the mother were of the anxious depressed type; in the daughter they were more katatonic in their general character; In family 7 paranoid states were present more in the descendants. while in family 8 it showed in the ascendants.

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[25] Ueber die Vererbung endogener Psychosen in Beziehung zur Classification, Monatschrift f. Psych. und Neurol., 1901, ix, p. 161.

[26] Traité des maladies mentales, 1860.

[27] Lehrbuch, loc. cit.

[28] Arch. f. Psych, 16.

[29] Monatschrift f. Psych. u. Neur., 1900, 7, p. 127

In 6 families showing senile dementia in the ascendants, in 5 descendants dementia præcox appeared. Idiocy was a result in the sixth. Further he shows that in 29 cases of dementia præcox occurring in brothers and sisters, 26 showed a similar inheritance in the parent, and 3 a dissimilar. The similar inheritance was manifest in 22 families with dementia præcox and 4 families with manic-depressive insanity.

Sioli's[30] excellent though now somewhat outdated study points in much the same direction. In his family 5, Schumann, it would appear that one of the descendants suffered from katatonia, another from manic-depressive insanity, while the ascendant had a manic-depressive psychosis. In family 6 a dementia præcox followed an alocholic insanity. Family 7 shows a probable dementia præcox following an uncertain psychosis. Family 11 shows well-marked dementia præcox in ascendant and descendant, family 14 and 15 the same picture.

In some particulars the hereditary element in the katatonic alliance seems to show a variation. It is of particular interest to note that the founder of the group of katatonics, Kahlbaum[31] says, p. 54, that heredity plays little part in the causation of katatonia, and in his 50 cases in only 4 was a definite history obtainable, while in all of the other cases no mention was made of it, and in the greater part of these it could be definitely excluded. Pronounced characters were, however, found in a greater number of instances—as high as 11 in the series of 50 cases. Other studies on katatonia and heredity are not accessible. While our present interpretation of the katatonic syndrome has undergone some modifications, it seems that of all the presently recognized approximations, katatonia differs in this important respect.

My own observations on cases of well-developed dementia præcox which I have been able to follow for many years and whose parents have been well known to me, has shown that three elements have been most emphatic in the ancestry: dementia præcox itself, alcohol, and abnormal personality or crankiness, if I may so express it. Alcoholic

[30] Ueber directe Vererbung von Geisteskrankheiten, Arch. f. Psychiatrie, 1885, xvi, 113.

[31] Die Katatonie, oder das Spannungsirresein, 1874.

parents have, in my experience, been most responsible for the hebephrenics; in many instances the alcoholism has been a symptom of profound neurasthenia or even a dementia præcox at a more advanced age, to which it in turn has contributed, and established a vicious circle which has left its impress in heredity. Pathological characters have constituted another most striking feature in my series of cases. These have been psychoneurotics in verity, and in this respect I agree with Tonnini—since these people may well be termed cranks. Marital incompatibility, due to the inability of one or both parents to adapt themselves to common-sense relations, has been a significant factor in many of the paranoid dements that I have been able to know well in the pre-dementia stages. This has had a marked influence on the education as well—which point properly belongs to the subject of our inquiry. One other class may be termed the "derelicts"—themselves in all probability suffering in some slight degree from dementia præcox coming on later in life. Many of these group themselves in the alcoholic class, since with the advent of manhood they sink back in the struggle for subsistence and are unable to compass more than small clerical positions. Many of these are the semi-failures of life—those pushed aside in the struggle, and forced to be content with small returns. This class has contributed the greater number of all classes of precocious dements to the population in my experience.

As to the influence of inbreeding, experience has shown that it all depends on those that breed. If neuropathic trends are present in those of blood relationship it is much more likely than not that the children will suffer. Healthy stock intermarrying often shows excellent progeny. Woods[32] shows that the usually accepted beliefs regarding royal intermarriage are in need of more explicit statement, and as now stated are more usually false than true. Thus it seems from his analysis that close inbreeding of the Hohenzollerns produced no bad effects until insanity was directly introduced into the stock, when the evil results began to appear in Isabella, Queen of John of Castile, and later inbreeding caused great havoc with the stock. The effects of insane stock appear very strongly in the Hapsburgs. The general influences drawn, however, accentuate the first statement of the position that inbreeding is bad only when the characters bred are in themselves distinctly pathological.

When, however, the pathological commences to heap up as has been noticed in the Hapsburgs,[33] then a distinctly degenerative stock results, and inferences regarding the probable outcome in such families have almost the value of prophetic insight. In many marriages one may say that adolescent breakdown in the progeny is foreordained.

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[32] Heredity in Royalty.

[33] Kekulé v. Stradonitz, Ueber die Untersuchung von Vererbungsfragen und die Degeneration der Spanischen Habsburger, Arch. f. Psychiatrie, 35.

Peipers,[34] who has contributed one of the most thorough of recent studies on this subject, believes that we are not yet in a position to draw any positive conclusions. His position seems to favor the belief that the marriage of blood relations is not attended with the serious results that have been laid down.

What is needed in the analysis of heredity is more than we have been in the habit of recording. We have been too much concerned with spinal-cord disease, apoplexies, etc., as hereditary factors, when they often may have no relationship whatever to the psychoses in question and might be neglected in our judgment of hereditary factors. A closer and more detailed account of the special personality of the ancestry is of more value than the vague terms, family neuropathic, mother, father, or grandparents insane.[35]

It seems almost futile to assert that insanity is not an entity. Everyone knows it, yet the phrase is continually in use as though it represented a stable entity; we have rather a host of shifting conditions, and our mental histories will be all the more valuable if more specific descriptions and fewer generalizations are recorded.

Alcoholism, vagabondage, ungovernable tempers, peculiar characteristics, great emotionalism, often seen in exhorters, etc.,[36] tendencies to antisocial conduct, to suicide, to thieving, to deceit, etc., these are all in need of recording, without undue emphasis, however, if a better history of heredity is to be written.[37] [38] Even then evidence is not lacking that in many directions we are building on shifting sands,[39] especially when we read in the paper of Jenny Koller[40] that the healthy-minded people show mental heredity in 59 per cent. of the population investigated.

Do the so-called physical stigmas assist us in forecasting a possible adolescent breakdown of the type here discussed? Purely in a most general way. While there is no doubt that one will find among our institutions a greater proportion of such stigmas, correlations needful for our present inquiry are not yet available. The modified psychiatric teachings will perforce render the older teachings only partly applicable.

A second factor that remains to be examined is that of tire, or fatigue. It would be unnatural to find a concordance of opinion with reference to the influence of fatigue on the nervous system of adolescents. The vehemence and almost violence, one might say, of those who advocate reduced educational schedules on the ground of their inducing excessive fatigue in children and young adults, with consequent nervous and mental reactions, has its counterpart in the extreme position of the skeptics who claim never to have seen any results of intellectual fatigue. It is more than probable that the natural adjustments of the normally formed adolescent to the influences of fatigue (laziness, distraction, etc.) will prove sufficiently protective, yet for the adolescent with some of the hereditary factors already outlined it is a fact the significance of which cannot be controverted that fatigue is a highly important element in their mental breakdown. So that while some may fail to be impressed by some of the many carefully conducted psychological studies into the influences of fatigue, these studies nevertheless show in a graphic manner that which clinical experience has amply demonstrated. School work is not the only cause of fatigue by any means, but it plays a rôle in the genesis of the neurasthenoid background which is so prominent a feature in many of the pre-dementia præcox signs, and one cannot fail to be impressed by the unnatural fatigability of these individuals.

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[34] Consanguinitat in der Ehe und deren Folgen fur die Descendenz, Allg. Zeitschr. f. Psychiatrie, 58, 1901, p. 793.

[35] Diem, Arch. f. Rassen und Gesellschaftsbiologie, 1905, Heft 182.

[36] Nacke, Allg. Zeitschrift f. Psychiatrie, 63, 1906, Heft 3, 4.

[37] Strohmayer, Ziele und Wege der Erblichkeitsforschung in der Neuro- und Psychopathologie, Allg. Zeitschr. f. Psychiatrie, 61, 1904 p. 355.

[38] Individuelle Geistesartung und Geistesstorungen, Th. Tiling. Grenzfragen des Nerven und Seelenlebens 1904, No. 27.

[39] Lorenz, Lehrbuch der Gesammten wissenschaftlichen Genealogie, 1898.

[40] Arch. f. Psychiatrie, vol. xxviii.

Such good observers as Emminghaus, Ziehen, Eulenberg, Pick, L. Strümpell, Kahlbaum, Hecker, Christian, Wille, Uffelmann, Binswanger, Baginsky, not to mention scores of others of equal prominence, all describe well-marked neurasthenic developments in the child and young adult, and ascribe them to the overburdening of school life. Among the ancients Willis says, in his striking way, that which I have quoted in my introduction to this paper. Kahlbaum, in his celebrated monograph, writes that he has been struck with the fact that so many teachers, sons of teachers, theological scholars, have been affected by the katatonic process. He believes that intellectual strain is one of the foremost of the contributory causes to this type of breakdown, the sexual life also aiding.

Intellectual overstrain is considered by him as of very practical importance, so much so that he says (p. 56): "Dass es, 'die intellectuelle Anstrengung' vorzugsweise die Ueberanstrengung beim Lernen ist, welche die Katatonie eigenthümlich ist."

Illberg, in a recent monograph, says that the great majority of his patients were hard-working and industrious. One-half were well endowed; the others less so. In those of less marked ability a protracted mental overstraining in the years of development is not without etiological significance. Hecker had called particular attention to the backward ones who were unable to keep up in their development, and Heller[41] has dwelt particularly on the psychoses of these inferiors. But the more striking instances are those who have been precocious, and attention should be carefully focussed on the precocious children, a great many of whom have suffered from dementia præcox. Baur[42] goes so far as to say that all mentally precocious children, so called in ordinary life, are pathological, and he believes them mentally ill, and are called on too early to endure great fatigue. These irritable, weak, wonder children are a veritable fata morgana, and are too often called upon to exhibit their striking qualities. If such children are not to be offered up to the altar of their parents' pride, they must be denied routine school exercises and be carefully taken care of rather in the playground. To stimulate such children to wonder-working stunts for proud aunties is to pour oil on the fire and hasten their breakdown. "Many of these wonder children are often only straw fires—they burn brightly and fiercely for a time and then all is over."

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[41] Ueberburdungspsychosen bei minderwertigen Kindern, Zeitschrift f. Schulgesundheitspflege, 19, 1905, p. 649.

[42] Zeitsch. f. Schulgesundheitsplege.

The symptoms that develop in these children as a result of overwork plus other factors concerning which we are unable accurately to estimate are very striking. Certain forms of excitability and restlessness should be distinguished from the ordinary restlessness and general tire states that are normal in all adolescent school-rooms. The pathological tire is one that does not recover as a result of the usual resting period, and these children are often the very ones that do not get the resting period because of a certain intensity of their application to their work or to reading.[43] The adept teacher recognizes the period of oncoming fatigue by the marked increase in mistakes and the flagging attention. School anxiety, particularly in starting new work, is to be carefully studied, either as a sign of fatigue or as a physiological negativism to which fatigue has given birth as its corrective. Changes in the mood are of much moment. Sudden rudeness, excessive selfishness, irritability and peevishness, lowery and quarrelsome, these often indicate more than the normal amount of fatigue, and are often the precursors of frank mental tire.[44]

Prominent among the more intellectual process disturbances, particularly in the neurasthenoid states, are memory defects, both for objects of sense as well as abstract concepts, although the more complex memory pictures seem to be more hazy. There is slowing of the mental processes and a subjective mixed sense of inability to get to work. The attention wanders, and the adolescent finds it difficult to control the day dreaming. He becomes lonelier in his disposition, refuses companions, and takes long walks. Occasionally one finds these hard-working students walking in their sleep, and not infrequently they go about in the night hastily attired or even not dressed at all. Frequently one finds great anxiety on the part of the person himself not to be left alone at night lest he will do some such foolish thing. Out-and-out neurasthenic symptoms are by no means infrequent in many young adults as a result of their school work.[45]

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[43] Spitzner, Anzeichen beginnender Nervosität, Zeitschrift f. Schulgesundheitspflege, 16, 1903, p. 395.

[44] Hintz, Geistesstörungen unter den Schulkindern, Zeitschrift f. Schulgesundheitspflege 1899, p. 179.

[45] Emminghaus, Die psychischen Störungen des Kindesalters, 1887.

What constitutes this mental surrmenage does not permit of clear-cut definition. It shows more in its results than in the making, and the integer of most importance and least conspicuousness is that subtle constitutional background. The amount of mental work seems to play less role than the method of its acquirement. Children well trained from infancy seem to stand increasing strains without serious embarrassment, while those who have had little or no training and are suddenly called on for prolonged cerebral work show tendency to break down out of proportion to what might be expected.[46] Christian and others in France have made the observation that when an ill-directed ambition has stimulated children of psychically poor rural stock to take up intellectual pursuits in the urban centres, dementia præcox is not an infrequent result.

Whether we can make any comparisons that are valid with reference to the differences of surrmenage in urban and rural communities is open, in view of the enormous complexity of our social conditions, particularly the incessant fluctuation and going here and there in our cities. Gaupp,[47] who has given one of the few studies in this connection that merit serious consideration, says that one very characteristic difference between the receptions of a rural clinic (Heidelberg) and a city clinic (Munich) is the greater number of young patients received in the latter; possibly indicating one important fact at least, that of the greater mental strain in the city environments. As to the matter of the group dementia præcox itself, seen in all its forms, he says that they seem to be present in about the same number in both places, but that the city shows a great many more young cases, particularly those with marked katatonic excitement with relatively good prognosis, that is, as far as the individual attack is concerned—a fact corroborated by Dr. Gregory in his experience at Bellevue—and many more of the more prolonged paranoid dementias or perhaps Magnan's chronic systematized deliria, particularly in the female sex. Thus Gaupp's study would point in the same direction that the increased tensions of city life have an important bearing on the development of dementia præcox, and these tensions all involve greater mental competitive functioning which lead to neurasthenoid states. There is a great need for rest cures for large numbers of tired teachers which can be run on an economical basis, and for the "rest" places which Sommer has been pleading for for years in connection with our museums, galleries, etc. The fact that the needs for this class of "erholung" have been expressed by all speaks for a tacit agreement that mental fatigue is many sided—it does not always mean books,—and that any measures taken to minimize its effects will result in healthier mental activities.

This portion of the theme might be expanded, but the recognition of the influence of fatigue in bringing about lessened mental functioning is almost a common-place. The aspect of the question of most practical interest at the present time is for greater clarity of view as to the more essential phenomena of fatigue and their earlier recognition, and differentiation of the more and less important of these signs and their more definite connection with personality and its balances. The highly artistic symphonic concert that leaves one adolescent almost a wreck from overstimulation may put asleep another of different reaction tendencies, and the recognition that there is a necessity for the clearer insight into what may bring about fatigue in the specific individual is imperative if an enlightened prophylaxis is to be taught and brought from hazy generalities to clarified, even if partially inadequate formulæ.

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[46] Wille, Die Psychosen des Pubertsalters, 1902.

[47] Die klinischen Besonderheiten der Seelestorüngen unserer Grosstadtbevolkerung, Munch. med. Woch., 1906, June 26, July 3, pp. 1250, 1310.

Other occasional causes are many, but I have chosen to regard their discussion as being premature at this time. This pertains particularly to masturbation, concerning which much might be said, pro and con. The transcending influences of the sexual life are recognized by all. Masturbation is but one symptom (a sexual catharsis) in this large function, and the study of the cryptogamic development of sexual ideas, brought into scientific sanction by the methods of Breuer and Freud, Jung and Ricklin, Janet, and others, cannot fail to impress the student that what is so important as a disaggregating emotional influence in hysteria must play no small role in fatigue and even in the dissolution of dementia præcox. But for the present I prefer to neglect this factor, recognizing that a middle ground must be taken. It is the tendency for neurologists to look upon onanism as a result, rather than as a cause. To me it is far from being clear that it is not both in many instances.

PROPHYLAXIS. The trend of the point of view adopted leads to the assumption that an enlightened pedagogy can be of more than casual service in the prevention of mental breakdown. This belief is, I believe, abundantly well warranted. But its recognition also implies a restricted application. It were folly to believe in this century or in the next to come that mental deterioration may be entirely avoided by proper teaching. The very protest that is so frequent in the minds of those who object to the term dementia præcox on the ground that it seems to imply a hopeless prognosis for this group of clinical forms, whereas so many do get well, is a sufficient indication that an abnormal functioning, which is self-limited in many instances and tends to recovery if more or less left alone, can be radically improved by earlier recognition of its fundamental character and the institution of proper physiological correctives.

Teachers are born, however, and not made. Pestallozis and Froebels are as infrequent as Dantes and Goethes, and the applications of pathological pedagogy are in a primitive stage, apart from the tact and insight of the born teacher or the wise parent.

On a priori grounds it is self-evident that the children of abnormal parents should be brought up with more than the usual amount of care, particularly during the period of puberty and early adolescence. What that "unusual amount of care" really consists in in specific terms does not permit of exact description. We as physicians are called upon to exercise that rarest of gifts, clinical tact, in molding the details of daily life and the modification of the home atmosphere to suit the abnormal child. To do this one often has to go to the earliest years of the child's life and obtain as complete a record as possible of the personality. This opens up the entire problem of the nervous child—here touched upon only in its most general features.

Nervousness in very young children is by no means infrequently observed. Even in the first months of life and among nurslings it shows itself as well. Römer[48] has called particular attention to this subject as occurring in nurslings. In older children one of the first signs of nervousness is disturbance in sleep.—Oppenheim.[49] This shows itself in a variety of ways, but is of particular moment in those in whom yawning and rubbing of the eyes is present, and they are distinctly tired and sleepy. On being put to bed, however, they are soon wide awake, are laughing and playing and become distinctly excited and then exhausted, have a short, restless, unsatisfactory interval of sleep. This should be carefully differentiated as an etiologlical factor from the same phenomena which may be noted after an overloaded stomach, or due to the presence of worry, or to a late and too violent or prolonged romp, or after the excitement of late travel, of hearing unusual street noises, or seeing sights, a parade, a fire, etc. Restlessness in sleep may often be complicated by definite night terrors, and with definite convulsive seizures, often epileptiform, accompanied by cries and apparent hallucinatory influences. Some of this type of neryousness could be avoided if the children's minds were not excited by tales of a weird and terrifying nature—often told by older brothers or sisters, by servants, or even by parents themselves. The carrying over of the feelings and thoughts experienced in the nightmare to the daily wakened period is of special moment.

Further signs of the nervous child are not to be overlooked. While they cannot be more fully developed at the present time, special stress should be laid upon the observation of attacks of sudden anger and passion, coming often out of a clear sky. Excessive reactions to emotional causes, such as blanching of the child on seeing an accident or during the recital of an exciting passage, the tendency to cringe and hold back on starting to cross a crowded street, etc., are signs of such nervousness. Tic-like movements of the face fingers, or body are to be noted, and special observation taken of the children who are never happy unless surrounded by a store full of toys and who play a few moments with each and are continually dissatisfied. Nervous eye symptoms, asthenopias, should not be overlooked.

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[48] Die Psychopathischen Minderwertigkeiten im Säuglingsalter.

[49] Nervenleiden und Erziehung, 1899, p. 47.

The tendency to exaggerate the importance of eye anomalies now so prevalent should not, however, permit one to adopt a *laissez faire* policy. These are among the more important very early physical signs of a nervous child—signs that indicate that these children are worth observation on the part of the pediatrist and the neurologist.

Equally worthy of record are the mental signs of anxiety as a neurasthenic sign in children, which may develop true phobias or impulsive ideas, even imperative ideas and imperative actions. The children who talk about jumping into the water or jumping out of high windows, or wanting to stick a sharp instrument into a brother or sister, those that show impulsive desires to steal, to hoard great quantities of things of a trivial nature, show these imperative modes of action which are signs of abnormal if not unhealthy mental functioning.

Given such indications of the past history and the development of the neurasthenoid states, with other of the phenomena already recorded, our problem is not a simple one. If to these are added attempts to set fire to buildings, strong sexual excitement leading to exhibitionism, the somnambulistic tendencies already spoken of, and the proclivity to continual running away from home, the situation takes a serious attitude. Stress should, however, be laid on the fact that one must not draw conclusions relative to the psychopathic character of the child on one or more signs, but the entire history needs to be carefully weighed.[50]

Inasmuch as a psychological negativism is present in so many of these pre-dementia cases, one of the first questions that arises is, How shall these resisting, self-willed, often cruel or tantalizing young people be corrected? "Spare the rod and spoil the child" is the old maxim, but to apply the rod has its dangers, and is of particular menace in certain types.

The irritable, resisting, and suspicious child is harmed by flogging, almost more than any other type. Any brutal method of punishment for his offensive acts or manners drives him more and more within himself. Suspiciousness becomes generalised. It passes beyond the known source of ill-treatment and begins to include other possibilities, and thus lays the foundation for a paranoid coloring to daily life. Normal natural resentment finds an occasional outlet in sudden, violent outbursts, at times with almost maniacal vehemence, often with alcoholic or sexual accompaniments and disastrous after-results. The intractability which should have been overcome by persuasion or by the careful teaching of cause and effect develops by the flogging method into more than simple resistance. It becomes aggressive revenge, and the entire affective life takes on a negativistic antisocial attitude. This forecasts a struggle with his environment on the part of the individual, and often leads to his ultimate passing into the ranks of the insane, or, if the mental breakdown is not too early or too marked, into the criminal fraternity.

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[50] Koch, Die psychopathischen Minderwertigkeiten, 1891.

Of all reformatory methods, the "big stick" one is the most dangerous in such unstable personalities. With inevitable fatality it drives the boy from home to vagabondage, bad hours, vicious company, and precocious dementia. It is true that the parents' plaint that they can do nothing with such a born vagabond is often only too well founded in fact, but there are many who in early childhood could have had the negativistic tendencies carefully pruned before they choked the entire mental life of the adolescent. The difficulty in such cases usually lies in the fact that the corrective measures are too long delayed. The parents, through no fault of their own, save ignorance, are not able to read the signs and avoid the disaster.

The girl of emotional instability, if unduly stimulated by late hours, too much excitement, often "goes wrong." Her parents' surprise and horror, coupled with their recriminations, drive her from home to become a prostitute. It she possess personal qualifications beyond the average she may live in comfort, and thus forfend the day of disaster, but as a rule, she forms one of a large number of her kind; disease and excitement strain her unstable emotions beyond endurance, and early in life she loses her mental balance. A large percentage of precocious dements among women have just such a history.

Some of these could undoubtedly have been saved by sympathetic and wise treatment at home. If the saving process has not been begun in childhood and the early teens, by regular hours and interesting occupations, the emotionally high-strung girl who has slipped, or who has had some indiscreet adventures, may still grow into a normal woman by their help. The removal of the family to another town, a visit, or travel, to bring new companionship, and the cultivation of new tastes and interests, will often give a girl self-control and respect for a well-ordered mental nature; whereas the slightest invective or harshness will make her sever all her old relations. The narrow path of wisdom that chooses between firmness and overstrictness, between indulgence and sensible sympathy, is a hard one for many parents to find, especially when the uncontrolled emotions of their daughters are the natural sequence of their own lack of control. Nevertheless, the parents or guardians are the responsible ones if in the period of adolescence a girl who is merely excitable and emotional is allowed to run the course which leads to mental destruction.

Probably the most important element in the prophylaxis of dementia præcox for the nation is the forbidding of marriage. The children of such patients almost invariably bear a taint. Nevertheless, the physician seldom has the opportunity to exert his influence: for parents are not in the habit of consulting a specialist to ask whether a hysterical, excitable daughter ought to marry, or whether the ungovernable temper of a son means mental inequilibrium. The parents hope that the settling-down process of marriage will eliminate unpleasant characteristics, never dreaming that the existence of a certain half-dozen symptoms would indicate to the trained eye the inevitable breakdown of dementia præcox. Could parents, and even the young people themselves, clearly foresee the suffering that would come when the mental decay became manifest, when the husband became a charge rather than a wage-earner, they would agree that it were wise to think twice before consenting to marriage.

When the physician is consulted, the problem of diagnosis requires particularly keen judgment. I have personally known a woman who remained unmarried until fifty, because her physician hinted she might break down mentally, but who eventually, after a sane, keen-minded life, became the second wife of the man who had first wooed her. Her physician's ill advice caused her unnecessary suffering because he mistook nervous tire for a mild psychosis.

On the other hand, it is known that alienists have advised young women to marry, on the ground that sexual completeness will allay their irritation and cure what had been diagnosticated as hysteria. Later when well-marked katatonic signs with dementia have appeared, they have realized that the breaking up of the home and the presence of tainted children have been the direct result of a mistaken diagnosis. Special stress must be laid upon the differential diagnosis of those presenting hysterical symptoms. The so-called hysterical stigmas, such as anesthesias, slight paralyses, etc., are of little value, however, in the differential diagnosis of hysteria from a malign psychosis.

A sharp distinction should be drawn between hysteria and hysterical. Hysterical symptoms are common to all of the psychoses, and, as is well known, are met with in great profusion in the organic nervous diseases. Their presence should not mislead one into making the diagnosis of hysteria, which is a fairly definite affection. Dream states resembling those of hysteria, when accompanied by echopraxia are strongly indicative of katatonia; indeed, Trömner[51] says that this is an almost invariable differental. Command automatism, catelepsy, echopraxia, echolalia, speech confusion may each or all be present in hysteria, but in most of these, close observation, or the interjection of an affective state, will have more influence on the symptoms in hysteria than in dementia præcox, and in spite of the theatrical and impulsive conduct, the signs of mental weakness and primary confusion are more apt to be lacking in hysteria.

I cannot enter into the subject of the differential diagnosis of dementia præcox and hysteria more fully, notwithstanding its importance in this respect. Jung's recent work is a classical contribution to this subject. Particular emphasis should be put on the necessity of clearer differentiation of an early dementia præcox from neurasthenia and hysteria, especially in view of the problem of marriage. Manic-depressive insanity in its mild hypomanic phases is also to be borne in mind.

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[51] Jugendirresein, 1900.

To revert to the subject of pedagogy: the manner and method of education for these children, if helpful in warding off disaster, is of the utmost importance; but we are forced into the most general platitudes when called upon to express in abstract what constitutes an effective prophylaxis. If the questions concerning the general problems of education for the healthy are the legitimate field of so much active discussion, and even acrimonious dissension between pedagogues, surely the more intricate questions of the training of the abnormal child—I do not here refer to the weak-minded or imbecile child—permit of even wider latitude of discussion.

One of the first generalizations that crops up is that these chlidren should lead a simple outdoor life. But, on close scrutiny, of what does this consist? My own observation confirms my belief that this method, as usually practised, is distinctly harmful, for the usual idea underlying its application is that the air will do everything. The difference in air in city and country is a comparatively negligible quantity, and if, added to the change to the country, one has bad cooking, bad roads, poor schools, and poor companions, the "simple life" may prove a hideous mistake. Yet a country life with proper care and surroundings is preferable. Even a simple country school, with few scholars, may be safely chosen, if the teacher be wise and firm. But in the general run, books should be neglected for occupations in the nature of manual and muscular training. I believe very firmly in the teaching, particularly of these queer children, of the small conventions of life, at a time, especially, when they are very naturally acquired. These are the oil of social intercourse, and their early acquisition often saves that embarrassment and even mortification which so frequently follows brusqueness and boorishness, and which leads to brooding and a churlish, solitary, self-centred life.

Sports are highly desirable, but they should not have too strong an element of competition in them, nor should they be advocated to the point of excessive fatigue. Rowing, skating, swimming, and horseback riding are to be classed in the front rank.

Education through the medium of industrial occupation is especially useful. Statistics are not available to show that workers with the hands—carpenters, wood workers, iron workers—are free from dementia præcox, or that these occupations are free from the results of early breakdown, yet the universal experience is that brain workers suffer the earliest, if not the most. Therefore, I feel that industrial training is a desirable mode of occupation for many of those who are on the brink. Those sanatoria in which the arts and crafts are taught are of great benefit for many of these young people.

Competitive intellectual pursuits are to be carefully avoided. The working out of complex themes, steady composition, much arithmetical calculation are undesirable. Debating, memorizing, and dramatics I consider harmful. In the choice of subjects of study one should avoid the larger problems, that is, those of greatest complexity. The study of sociology has proved, in my experience, the brightest and most destructive flame which has attracted the poor præcox mind. It is a very usual experience to find the paranoid præcox patients striving to understand the unknowable with absolutely not a single fact of the elementary sciences.

To interest these young people in natural objects is highly desirable, but the gamut of the teacher must be wide. The ordinary teacher of science who has mere book knowledge is a hopeless guide. One needs the old-fashioned naturalist tendencies to make the open air interesting and instructive to pupils who have so little tendency to become objective or to see things about them in daily life.

By virtue of the growth in wealth in this country, and the wide dissemination of country homes, with their fully or moderately developed farming addenda, I believe that many young people could well choose the better understanding of agricultural pursuits to the advantage of their mental health. I personally know of a number of men who, recognizing their bad mental heritage and perceiving while still young the strain of urban competition, have changed their mode of life entirely, have taken up agricultural pursuits and are now after twenty years well, strong, and happy, and some even have acquired more than a modest competence. The agricultural courses in our colleges should not be overlooked in this connection.

For the more careful direction of many of these individuals, who unfortunately are left to drift past the time for reconstruction, there is no doubt that specially trained individuals are needed—individuals whose point of view is the understanding and correcting of unhealthy mental habits, life habits of thought which when set show the dysharmony of the individual with his actual surroundings. We have all seen a number of these teachers as they now exist. We may term them tutors or companions or trained nurses, as we will, and employ them to look after abnormal adolescents. Most of the failures that have come to my observation, where financial resources could have rendered the situation ideal, have in large part been due to the fact that the teacher or tutor or companion had been ill prepared for the special problem in hand, and has made a failure of his opportunities. There is a large opportunity for the teaching profession of this country to evolve the mental healer in this sense, otherwise even the crudities of the quackish upstarts will be welcomed by a distressed and despairing public.

THE SURGICAL TREATMENT OF GOITRE.

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The relatively infrequent occurrence of thyroid tumors in most parts of this country accounts in part, no doubt, for the tardy acceptance of operative treatment. A few sudden deaths from fulminating appendicitis or ruptured extra-uterine pregnancy soon convince the profession and the public of any community of the necessity for prompt and radical treatment. On the other hand, the slow growth of a goitre, the gradual development of alarming symptoms, and the small number of cases seen, have made all concerned ready to temporize with useless or even dangerous palliative measures, in spite of the fact that the ultimate outcome in many of these cases is certainly fatal and in almost as large a proportion of cases as with the more acute abdominal diseases just mentioned. In Switzerland, where goitre is a common disease, partial excision of the thyroid gland is as common as appendectomy is in the United States and is more free from danger. Kocher of Bern[1] reported 2160 excisions, with but three deaths in his second series of 1100 cases. Since 1902 the number of operations has increased so rapidly that he has since reported 3000 excisions. At the German Surgical Association last spring he reported that one of his assistants had operated upon 661 patients with but one death.[2] Probably no surgeon has had as great experience as Kocher, but in almost any Swiss clinic the operation for goitre is a daily occurrence, the results are highly satisfactory, and in practically all Europe the surgical treatment of goitre has been for many years a well-established procedure. As compared with statistics of European countries, the number treated in any American clinic is ridiculously small. Indeed, many American surgeons have never done a thyroid excision and apparently many, even among our most progressive practitioners, have never heard of surgical treatment for goitre, or if they have, do not consider it safe to advise for their patients. Although goitre is much less frequent in this country than in certain parts of Europe, there seems to be no reason why we should not offer our patients as satisfactory surgical relief from this distressing condition as is given in other civilized lands. Before discussing this question further it may be worth while briefly to consider the varieties of goitre and a few facts regarding the causes, symptoms, and diagnosis.

Thus far I have used the term goitre in the sense generally employed, meaning any enlargement of the thyroid gland, but these thyroid tumors may be divided into several distinct groups, with widely differing symptoms. Any extended discussion of the histology or pathology of thyroid lesions seems out of place: interest lies mainly along the lines of diagnosis and treatment. Enlargement of the thyroid may result from inflammation, hypertrophy, or tumors. The acute and chronic inflammations of the thyroid are of infrequent occurrence, and the symptoms of inflammation are generally readily recognized. The most important tumors are the various forms of adenoma, carcinoma, sarcoma, and cyst.

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[1] Chirurgische Operationslehre, Jena, 1902, fourth edition, p. 205.

[2] Annals of Surgery, 1906, vol. xlv, p. 758.

Simple and exophthalmic goitre are examples of the hypertrophies. The differences in microscopic structure of these hypertrophies have practical significance, hence, I mention that in simple goitre the enlargement is seen, by microscopic examination, to be caused chiefly by dilatation of the acini with colloid material; in exophthalmic goitre, on the contrary, the colloid material is lessened or even disappears, while there is a process of folding of the wall of the acini, giving increase in size of the gland and a larger surface of epithelial lining. Thus in exophthalmic goitre there is a very greatly increased area of secreting surface; the excess of thyroid secretion giving grave general symptoms. In simple goitre there is slight if any increase in secreting surface and pressure of the contained colloid material may even reduce secretion.

These various forms of thyroid enlargement cause various symptoms, but from whatever cause pressure symptoms are marked from the growth of a tumor of this important organ. Pathological examination is the true basis for classification of these as for other tumors, but we have to deal with three groups clinically: First, the group of tumors in which the symptoms, if any are present, are dependent solely on the enlargement of the gland and pressure on surrounding organs. Simple goitre, adenoma without metastasis, and simple cyst, while differing radically pathologically, all usually produce their symptoms in this way, by pressure only. In the second group we may place the malignant tumors, whether carcinoma, sarcoma, or adenoma with metastasis; these give the signs and symptoms of malignancy, in addition to the pressure symptoms. A third and most important clinical group is that accompanied by pronounced general symptoms, the exophthalmic goitre, usually called Graves' disease by English writers, and Basedow'sche Krankheit, by the Germans. In these cases the enlargement of the thyroid is accompanied by pronounced nervous symptoms, especially tremor, by protrusion of the eyeball, rapid heart beat and other vascular changes. It is important to note that nervous and vascular changes may exist without exophthalmos, or at first nervous symptoms alone may be present or vascular symptoms alone.

As little is known of the causes of goitre as of tumors in general. It is well known that they occur much more frequently in mountainous regions and are especially prevalent in Switzerland and the surrounding mountainous portions of Europe. I am told b medical missionaries from the mountainous districts of Asia that goitres are extremely prevalent there also and frequently attain enormous size. Goitre patients at Johns Hopkins Hospital usually come from the mountainous districts of Maryland, West Virginia, and Pennsylvania. Exophthalmic goitre frequently follows great nervous strain and has been supposed by some to be a purely nervous affection. Others incline to believe that the nervous symptoms are secondary to the diseased condition of the thyroid. A few observers attribute the disease to toxemia of intestinal origin. The inhabitants of no special country and no race are wholly exempt. It certainly occurs with greatest frequency in young adult females, and pregnancy and child birth apparently have some influence. A great deal has been written on the etiology of goitre, but so few positive facts are known that extended discussion at this time seems unjustified.

Symptoms. With the benign and malignant tumors of the thyroid and simple goitre, symptoms are frequently entirely absent, with the exception of enlargement of the gland. In simple goitre the gradual enlargement of the thyroid may be associated, in the early months and years, with slight nervous symptoms, even palpitation and tremor. For this reason the early stage of a simple goitre may be mistaken for Graves' disease, but in these cases exophthalmos is said to be always absent. When exophthalmic goitre has existed for some time the bulging of the eyes, tremor, palpitation, and extreme nervousness, associated with enlargement of the thyroid form a striking clinical picture. Most of us are familiar with the eye symptom pointed out by von Graefe: the upper eyelid fails to follow the eyeball on looking down; and Stelwag's symptom, the apparent widening of the palpebral slit. When enlargement of the gland becomes great, difficulty in breathing and swallowing, change in the voice, cough, and sometimes even bloody expectoration are noticed. These symptoms, associated with a large goitre, are much less likely to be dangerous than when the enlargement is comparatively small, for then infiltrating carcinoma should be suspected.

Early diagnosis of enlargements of the thyroid is a matter of great importance. Kocher, Bloodgood, and others, have emphasized the fact that it is impossible to tell whether a tumor of the thyroid is malignant or not in the early stages of the disease, and unless a malignant growth of the thyroid is removed very early the condition becomes inoperable. At the Johns Hopkins Hospital the proportion observed has been 10 malignant tumors to 52 benign tumors; almost 20 per cent. and so large a proportion that the danger of temporizing with any thyroid tumor is easily seen.

Treatment. The question of treatment is one of great interest and of the greatest importance to our patients. Osler[3] declares that medicinal measures are notoriously uncertain, and the multitude of methods which have been suggested for exophthalmic goitre gives sufficient evidence of failure. One observer insists on the importance of directing treatment to the circulatory organs or perhaps the heart alone; another to the sympathetic nervous system, or some part of the general nervous system; another to the intestinal tract; another to the thyroid gland itself; one uses strong, another weak electric currents. Recently organotherapy has attracted much attention. Serum has been prepared from dogs and calves whose thyroids were previously removed; the milk of thyroidectomized she-goats has been employed under the name rodagen; the blood of patients suffering from myxedema has been injected; guinea-pigs, rabbits, roosters, and geese have been experimented with and more or less enthusiastic reports of results obtained have been given. Perhaps the most promising of the serums is that prepared by Beebe[4] from the thyroid of two persons who died from exophthalmic goitre. Rogers[5] and others have obtained promising results from the use of this serum, although very alarming symptoms were produced in some cases and no improvement in several. In his latest report Rogers gives four deaths in 54 cases treated by serum. Rhein[6] after reviewing numerous forms of organotherapy, expresses doubt whether the results are more encouraging than those obtained by the usual well-recognized therapeutic measures. It would seem that in spite of all efforts patients still remain uncured and experimenters are still looking for something new as a specific for exophthalmic goitre. While the internists have been treating almost every organ in the body, from the intestinal tract to the thyroid itself, with all kinds of drugs, serums, and extracts, made from animals and men, dead and alive, applied locally and taken internally, a few surgeons have been excising a part of the thyroid with most brilliant results in case of simple goitres and benign tumors, and although the mortality has been higher in exophthalmic goitre, the results have, on the whole, been better than in those treated medically, particularly when the unfavorable condition of the patient operated upon is considered. Recovery is very rapid after operation in these cases. There is little or no postoperative discomfort; usually immediate slowing of the pulse and relief of nervous symptoms. All of my patients have sat up in bed on the third day after the operation and have been out in a wheel chair on the fifth day. The scar left is practically invisible and is so low down that it is covered by the collar. Ten patients have come to me for operation within six months, and the extreme gratitude of these and all previously under my care, for their relief from their sufferings, has been such as I have never seen in the results of any other operation.

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[3] Practice of Medicine.

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[4] Jour. Amer. Med. Assoc., 1906, xlvii, 661.

[5] Ibid., 665.

[6] Amer. Med., 1906, xii, 164.

It is true that these operations involve several special dangers and difficulties: when greatly enlarged the thyroid gland is always in direct contact with the great vessels and nerves of the neck for some distance; frequently the great vessels are considerably displaced from their normal relations; in 3 out of 10 of my own cases the growth extended down into the thoracic cavity behind the clavicle and sternum in close relation to the apex of the pleural cavity; in all cases the thyroid gland is friable and the vessels are thin walled, easily torn, and capable of giving terrific hemorrhage.

In no modern procedure has there been a greater difference in the results of operative treatment. Why should some surgeons report a death rate of 10 per cent., others a mortality of less than 1 per cent.? This leads me to ask if surgery of the thyroid should not, for the present, be referred to those who have proved their ability to operate successfully. Should not the surgeon undertaking excision of the thyroid be familiar with details of the normal anatomy of the neck and upper thoracic cavity, with anomalies, which are of frequent occurrence, especially in the arterial and venous supply, and with the pathology of the thyroid lesions most frequently met? Would it not be worth while for the surgeon whose mortality rate has been over 1 per cent. in these operations, or especially those who are undertaking the operation for the first time, to take the trouble to visit Kocher's Clinic, or that of some other surgeon with a record for success in this operation, and carefully note the methods used? If all men had given themselves such preparation I believe many fatalities would have been avoided.

The experience of a number of surgeons, as shown by the statistics I have quoted, proves that the mortality can be reduced to 1 per cent. or less in simple goitre operations performed under local anesthesia. In view of these facts I quite agree with Kocher who states, that "It is not justifiable for physicians to introduce and follow medicinal treatment for a long time, when from the very beginning it is evident that it can be of no use. So soon as the goitre becomes dangerous, when there is much difficulty in breathing, or inflammatory changes; so soon as there is the slightest suspicion of malignant degeneration, operation is indicated. Suspicion of malignancy is always present when a person over thirty years of age shows enlargement of the thyroid without any definite cause. Unfortunately the majority of malignant goitres, which are not so uncommon even in relatively young persons, almost always come to the surgeon so late that radical operation is impossible. Goitres which are growing in the direction of the thoracic cavity are also dangerous, and should be operated upon. All goitres which reach a considerable size should be removed, and especially when even in a slight degree symptoms of Graves' disease are present, operation is the best means of prepreventing the further development of this serious disease."

It is not my purpose to discuss methods of operation in detail, Such matters may be interesting to those who are devoting their attention to surgery, but they have little interest for the average practitioner. Personally, I always use local anesthesia and have found it entirely satisfactory. By its use time may be devoted to painstaking dissection, so necessary where many important anatomical structures are concerned, without the feeling that injury will result from prolonged anesthesia. The extreme discomfort caused by retching and vomiting after extensive dissection of the neck is avoided. Perhaps most important of all is the greatly reduced hemorrhage; everyone is familiar with the congestion of the face and neck which frequently accompanies the use of ether and can appreciate how much the overdistension of the veins in general anesthesia would add to our difficulties. I believe with Kocher, Witherspoon, and numerous others that the use of general anesthesia adds great danger in goitre operations. Witherspoon[7] states that all patients suffering from exophthalmic goitre are affected unfavorably by general anesthesia, whatever the nature of the operation, and whether the thyroid gland is handled or not. He mentions a case in which alarming symptoms followed the use of general anesthesia for curetment. The symptoms which he reports are similar to those generally considered the result of thyroid toxemia from squeezing the secretions of the gland into the circulation by handling during the operation.

The incision should be long. I prefer the transverse collar incision of Kocher, which with careful closure, leaves little scar, and that located so low that it is easily covered. In order to avoid scar I close the wound with the finest embroidery needle, using very fine horse hair as a suture material. Such careful closure requires much time, but if local anesthesia is used this is a matter of little consequence.

The applications of regional anatomy in this operation are numerous and important. All larger branches of the superficial cervical and supraclavicular nerves of the cervical plexus may be avoided by placing the transverse incision between their regions of distribution, thus making local anesthesia simple and satisfactory. In dissecting the flap upward to expose the thyroid, these nerves and their branches may be entirely avoided, for the nerves pass through the platysma near their origin. Both the nerve trunks and the skin they supply are lifted with the flap which includes the fascia beneath the platsma. I am not aware that this great aid to the success of local anesthesia has been previously referred to by any writer on this subject. The muscles attached to the larynx should be dissected carefully, divided near their upper attachment so as not to injure their nerve supply which enters the muscle near its middle, and after removal of the goitre, carefully sutured. This will prevent much deformity of the neck and disability in using the larynx. The dissection should be most careful. The superior thyroid artery should be secured first, then after freeing the goitre from its attachments, it can be dislocated from its bed and the inferior thyroid artery secured. All large veins should be isolated and tied, as far as possible, after the arteries are secured. If these precautions are followed the hemorrhage will be trifling. The capsule on the side toward the larynx should be separated with a small portion of the gland itself in order certainly to avoid injury to the recurrent laryngeal nerve. This should also be carefully avoided in tying the inferior thyroid artery. With the strictest aseptic precautions and careful observance of these details, I believe that the results obtained will be most satisfactory.

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[7] Trans. Amer. Med. Assoc., Section Surgery and Anatomy, 1903, 311.

The prognosis in thyroid tumors is more dependent upon prompt and proper treatment than in most diseases. In this connection we have to consider, first, the immediate operative mortality; and second, the prospect of permanent relief. As regards danger to life there has been great improvement in the results in recent years. In 70 operations for ordinary goitre before 1850, Kocher placed the mortality at 40 per cent. In 400 operations between 1850 and 1883, it fell to 15 per cent. while, since 1883, it has steadily fallen to less than 3 per cent. Kocher's own being 0.4 per cent. in simple cases. Dr. Finney, of Johns Hopkins Hospital, tells me that he has had no deaths from the operation, including his cases of exophthalmic goitre. Bloodgood reports no operative mortality and no recurrence following operations for cysts[8] in a large series of cases, and in 1 case of malignant tumor removed early[9] the patient has remained well six and a half years. The results of operative treatment vary greatly in the hands of different operators, but several surgeons have reported a mortality of 1 per cent or less.

Charles H. Mayo[10] says: "The public has learned that operations on goitre are not fatal as were supposed from the results obtained when operations were made as a last resort on patients suffering from this disease and in a moribund state. The fact is that the mortality attending the operation (excluding cancer and advanced cases of exophthalmic goitre) compares very favorably with other major surgery; and, in the hands of those experienced, with much of the so-called minor surgery." As regards the second question, the ultimate relief, there is every prospect of permanent cure except in malignancy or exophthalmic goitre. Wolff's statement that when one-half of the gland has been excised for hypertrophic goitre the remaining half always diminishes, is confirmed by Riverdin, Kocher, Mayo, my own experience, and that of others. In cases of exophthalmic goitre, not in extremis, the improvement following operative treatment has been very encouraging. At least 50 per cent. of the patients have been entirely relieved and in a large proportion of the remaining cases there has been great improvement. This is a far better showing than can be made for any or all of the other methods which have been given extended trial in the treatment of exophthalmic goitre. Let us compare the results in cases treated medically. Williamson[11] reports 24 cases treated medically with fatal termination in 6 (25 per cent.), improvement in 7, condition much the same in 3, and almost complete recovery in 7 only. Möbius[12] quotes statistics from five authorities and the estimate of mortality is from 10 per cent. by von Graefe to 25 per cent. by Charcot, in cases treated medically. Mayo quotes Ord and Mackenzie who tabulated 56 medical cases of exophthalmic goitre which had lasted five years or been fatal. There were 10 recoveries, 11 made incomplete recovery, in 13 there were considerable improvement, 4 were unchanged, and 14 (25 per cent.) were fatal. Others make more favorable reports. Osler says, "A certain proportion of the cases recover, but when the disease is well developed recovery is rare." These eminent internists give a much graver prognosis for cases treated medically than may be offered to patients treated surgically. No competent surgeon should have one-half as great mortality as they give. For my own experience, in 6 of the 16 cases who have recently consulted me I have advised trial of palliative measures. One of these patients, a girl just past puberty, is apparently improving; 4 remain about the same and there is certainly no urgent need of operation; in 1, after several months of treatment, as the growth had increased and the symptoms become more annoying, the patient decided to undergo the operation, the result from which has been entirely satisfactory. Thus I have referred back to the internist for treatment nearly one-third of my cases, yet in only 1 case has there been definite improvement. But what can be expected from medical measures in the case of an enchondroma so large as to cause great difficulty in breathing and swallowing, as in 1 of my cases; or in the case of a cystic goitre so large that the woman had been unable to swallow meat or other solid food for two years, as in another of my cases. The physicians who had charge of these women would have unhesitatingly advised removal of an ovarian cyst of half the size, causing far less distressing symptoms, or a breast tumor no less likely to be malignant. I am sure the question may be left to the judgment of anyone whether operation is not the only rational treatment for all benign and malignant tumors of the thyroid, as it is for tumors in other parts of the body.

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[8] Surgery, Gynecology, and Obstetrics, 1905, vol. i, p. 113.

[9] Ibid., 1906, vol. ii, p. 121.

[10] Jour. Amer. Med. Assoc., 1907, xlviii, 273.

In 10 of my operative cases, there was extreme discomfort, and in several, symptoms dangerous to life, yet operation had been previously advised in only 1, and in several cases the patient was advised against operation by prominent physicians. Can it be that the internists who had charge of these patients had attempted to follow the after-results of their treatment and realized the absolute failure of medical measures to give permanent relief? Is it not time that the results of excision of the thyroid in the hands of experienced men should be recognized? Is there any other operation of equal gravity which has been performed by any man with but one death in 661 cases, as in Kocher's latest report?

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[11] Brit. Med. Jour., November 7, 1896.

[12] Nothnagel's specielle Pathologie und Therapie, Band xxii, p. 65.

In closing, I would again emphasize the importance of early diagnosis, when in some cases there is still hope of relief by palliative measures. I would refer once more to the grave prognosis in all well-developed cases of exophthalmic goitre; to the difficulties of distinguishing benign from malignant tumors of the thyroid in the early stages of development; to the frequency of malignancy and the absolutely hopeless prognosis in malignant cases unless the growth is excised early, before a positive diagnosis can be made. I would again mention that reports of cases treated by medical measures alone show a death rate higher than those operated upon, and this in spite of the fact that usually only the most unfavorable cases have been submitted to excision; that operative measures have cured or relieved a proportionately larger number than any (I might almost say all) other treatments. In view of these facts it seems clear to me that all patients resisting the approved therapeutic measures should be urged to consult a competent surgeon.

TENDON TRANSPLANTATION IN THE TREATMENT OF CONGENITAL AND ACQUIRED TALIPES.

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The general surgeon has always been more or less dissatisfied with the treatment of deformities by means of apparatus, and, has, whenever practicable, preferred operative treatment, with its rapid results, to the slow and at times unappreciable progress which is attained by the use of braces. It is, however, chiefly owing to the teaching of Vulpius in Germany, and of Tubby and Jones in England, themselves orthopedists, that tendon transplantation has become a recognized form of treatment for paralysis. In this country it does not appear to have been very widely employed heretofore, and patients with infantile palsy have drifted about from one clinic to another, or, in rare instances, have grown from children to adults under the care of one individual. And though it cannot be denied that the treatment by apparatus is a great help, not only in increasing cess, injecting the solution into the spinal canal, making the injections low down in the sacral region toward the bottom of the canal. Whether the results obtained by these injection methods are purely to be attributed to the mental effect, or whether they act reflexly, one cannot say. In refractory cases this method might be tried.

Of the old-fashioned means of treating this disease there are one or two things which may be mentioned. The first is to place the child upon a simple non-irritating diet and to restrict the amount of fluid taken, particularly the amount of fluid taken after four or five in the afternoon. Secondly, in some cases raising the foot of the bed so that the irritable neck of the bladder is not quickly affected by the first urine which enters the bladder. Certain cases are improved by a large amount of rest and leading a quiet life. General reflex irritability may be caused by too strenuous a life, particularly long automobile rides and the like. I have seen on several occasions the simple directions to have the child stay in bed until it is ready to get up in the morning, and to lead a quiet life generally, result in cure.

In hospital practice a large proportion of the cases can be improved by a more or less dry diet and restriction of fluids. Where there is no other indication for treatment I have found the use of atropine to give better results in a greater number of cases than any other one thing in the suggestions made. To be of any service atropine must be given in full doses. In nocturnal cases a dose at five o'clock and at bedtime is all that is required. In cases occurring both during the day and night, the administration of the drug every three hours is to be advised. I usually prescribe a solution containing 1 grain of atropine sulphate in 2 ounces of water. Each drop of this represents approximately $\frac{1}{1000}$ of a grain, and ordinarily about as many drops will be required at a dose as the child is years old; but this is not the proper method of ascertaining the dose. Starting with one or two drops each dose should be increased one drop at a time until flushing of the face and neck occurs some twenty minutes after the administration of the drug. The dose should be diminished one drop, and this amount continued until the child has ceased urinating at night and for at least two weeks later, when the drug may be left off gradually, diminishing a drop at a time until one drop is reached, when it may be stopped.

It is hardly necessary to comment upon all the things mentioned in the table above. The chief object in writing this paper was to call attention to some of the things which have been published recently, and to call attention to the fact that there exists a small class of cases in which the thyroid insufficiency evidently causes nocturnal enuresis and which may be cured by the administration of the thyroid with great benefit to the general health and growth of the child.

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SOME APPLICATIONS OF THE CREHORE MICROGRAPH, WITH ESPECIAL REFERENCE TO THE RECORDING OF HEART SOUNDS.

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The purpose of this communication is to make a preliminary report of a few methods of using the Crehore micrograph[1] in the nerve to persist (in the upper extremity) for three months or more; and at the end of that time it wlll still be uncertain whether the paralyzed nerve will ever transmit stimuli received through its new anastomosis. The question of the extent to which the degeneration of the paralyzed muscles has gone, must also be considered in the attempt to restore their contractility by nerve impulses. In tendon transplantation, on the other hand, the field is limited only by the number of tendons available; the results are definite and prompt; and no danger exists of rendering the paralysis worse than before the operation was done.

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[1] Jour. Exper. Med., 1911, xiii, 616; ibid., xiv, 339 to 365, and 520.

In congenital club-foot it seems safe to allow a longer period to elapse before resorting to tendon transplantation. Here, too, it is probable that correction of the deformity and maintenance in the corrected position, will, by facilitating the normal action of all the muscles, soon render the weaker groups powerful enough to hold the foot straight. If failure attends these efforts, and especially if maintenance of the normal position proves impossible, then resort to tendon transplantation will hasten a cure, and enable braces to be promptly discarded.

As preparation for the operation it is above all things essential that the foot be easily retained temporarily in the desired position, preferably in over-correction. This is to be attained by the usual methods of manipulation, stretching, and by tenotomies when necessary. When the foot has been made almost flail-like, so that its over-corrected position can easily be maintained by the pressure of one finger, then it is ready for the transplantation. It is best not to transplant at the same sitting at which a forcible stretching, under ether, is done. As a rule this procedure causes a certain amount of contusion and œdema, and would predispose an open wound to infection. Ten days or two weeks after the foot has become perfectly flaccid by stretching, the cast used for retention may be removed, and open operation performed. If necessary, of course, several stretchings are to be done before the final operation of tendon transplantation.

The technique of the operation is sufficiently simple, but requires an accurate anatomical knowledge, which is scarcely less important than the knowledge of the functions of the various muscles, on which the principle of the operation is based. The use of an Esmarch bandage considerably accelerates matters, since veins may be seen and avoided, and all the dissections are rapid and bloodless. The band should be placed *above* the knee, so as in no way to constrict the muscles it is proposed to transplant. It is needless to say that the surgeon must have decided positively on the method of operation before ether is given.

Periosteal implantation is generally acknowledged to give better results than transplantation into another tendon; and a more sure result will also be obtained by transferring a healthy tendon to the place of a weak one, than by suturing the distal end of the paralyzed tendon into a normal tendon. The typical operation is that for varus, where the peronei are paralyzed or weak, and where the tibialis anticus is the deforming factor. In such cases, therefore, separation of the tibialis anticus from its insertion, and its subcutaneous transference to the point of insertion of the peroneus brevis, with its suture firmly into the periosteum at that point, will give a much more satisfactory result than will division of the peroneus brevis some distance from its insertion, and the transference of its distal end into the tibialis anticus. More satisfactory results also will follow transplantation of an extensor tendon for extensor paralysis, or of a flexor tendon for flexor paralysis, than will attend the use of a flexor muscle to overcome extensor palsy, or vice versa. Yet when all of one group of muscles are paralyzed, it may then become necessary to transplant a muscle whose function is directly the reverse of that we wish to develop. Thus in Case II, in which there was paralysis not only of the anterior tibial group but of the peronei and of the tibialis posticus as well, I was forced to take the flexor longus hallucis, which was strong and a deforming factor, from the sole of the foot, and bring it forward through the interosseous membrane to the dorsum of the foot where it was sutured to the extensor longus digitorum.

The incision to separate from its insertion the tendon which it is desired to transplant need not as a rule be long. It is made in the line of the tendon; the sheath is opened, the tendon is grasped by clamp forceps to keep it from retracting when cut, and it is then cut at the desired point. The tibialis anticus must be shaved from the scaphoid and internal cuneiform bones, as it is none too long to reach to the insertion of the peroneus brevis. Longer tendons may be cut higher, and their distal ends sutured to neighboring sound tendons, as in the case of the extensor longus hallucis, whose distal end may be sutured to the extensor longus digitorum. The next step usually is to bare the tendon above the annular ligament, and draw it out here. Then an incision is made over the desired point of insertion, and by burrowing a subcutaneous channel up to above the annular ligament the tendon is caught in forceps, or attached by sutures to a probe, and drawn down to the point where it is to be permanently anchored. The incision over this latter point should not be made precisely in the line in which the transplanted tendon will pull, since the tendon might delay union of the skin by tension. It is better to make an incision oblique, or a little to one side, or even to turn up a flap of skin if more room is required.

The suture material is a matter of some discussion. Some surgeons think silk is preferable, because it is permanent, and hence there is thought to be less danger of the tendon pulling loose. Having had precisely this experience, however, with the patient in Case IV, in whom silk was the suture material employed, I feel rather partial to chromicized catgut, which has given perfectly satisfactory and thus far at least perfectly permanent results. The transplanted tendon should be sutured with the utmost possible tension, and with indubitable security. A fairly heavy needle and thread are required, and mattress sutures are the best. The knot should never be on the tendon, but is preferably placed to one side or other. In some cases, as in suturing the tibialis anticus to the tuberosity of the fifth metatarsal bone it is possible to place the knots in the sole of the foot, suturing through the bone—which is cartilaginous in children The knots should not be in any position in which they may subsequently cause annoyance. This applies especially to silk, which is not absorbed. In the sole of the foot they are protected by so thick a pad of fat that no trouble need be expected.

The skin wounds are closed with chromic gut, and the foot is dressed before the Esmarch band is removed. The limb is kept elevated both while the plaster is being applied, and for forty-eight hours subsequently in bed.

The cast may be changed at the end of three or four weeks, if it seems desirable to do so; but free motion of the foot should not be allowed for six to eight weeks. It is exceptional then for a brace to be required; but in some cases of extensive paralysis it may be advisable to continue the support to the foot for some months. Usually an ordinary shoe may be worn, or one with the sole raised on the transplanted side, the outer side raised for varus, the inner raised for valgus deformities.

For permission to operate on and to report the following cases I am indebted to Drs. G. G. Davis, and R. H. Harte, in whose services at the Orthopedic Hospital they occurred.

CASE I.—Israel S., aged seven years, entered the service of Dr. Davis at the Orthopedic Hospital, January 23, 1906. He had been born with marked equinovarus of the right foot. Until the age of four years he had been under treatment at another hospital in this city, where tenotomies of the tibialis posticus, tendo Achillis, and plantar fascia were done. The fact that the posterior rather than the anterior tibial tendon was divided shows the extreme grade of deformity present. Since the age of four years he has worn a brace, but has not been under surgical observation, the brace being repaired by the maker from time to time. When first seen at the Orthopedic Hospital, there was still present marked varus, the equinus having nearly disappeared. The foot was forcibly stretched the patient being anesthetized, February 2, April 27, and May 17, 1906. In spite of complete over correction of the deformity, the peroneal muscles remained powerless to hold the foot in the corrected position, and tendon transplantation was determined upon. On June 15, 1906, the tibialis anticus was separated from its insertion into the scaphoid and internal cuneiform bones, and by means of a fine-pointed bistoury the sheath of the tendon was slit up through the annular ligament without making a separate wound above this latter structure to draw the tendon out. An incision was then made over the tuberosity of the fifth metatarsal bone, and the tendon was transplanted subcutaneously across the dorsum of the foot from one wound to the other. By placing the foot in the position of valgus deformity the tendon of the tibialis anticus could now be made to reach the desired point, with great tension. By means of a strong curved needle, entered on the plantar aspect of the metatarsal bone, a mattress suture was inserted through the transplanted tendon, which was thus firmly pulled down and united to the periosteum, the knot being placed on the sole of the foot, and the suture passing through the bone. A second suture of chromic gut united the transplanted tendon to the tendon of the peroneus brevis, just at its insertion, and a third suture was passed through the fascia and periosteum on the dorsum of the foot. The skin wounds were closed with chromic gut, and a plaster cast was applied. The time of the operation was thirty-five minutes. The cast was removed July 20, five weeks after the operation. The transplanted tendon could be easily seen and felt beneath the skin in its new situation, and by its contraction effectually prevented the previous varus deformity. Four days later the patient was allowed to walk. To prevent any undue strain on the foot, he was directed to have the outer side of the sole of his shoe extended and raised a quarter or three-eighths of an inch. An instep strap was also applied. Fig. 1, from a photograph made March 5, 1907, shows the shape of the foot at that date. As he still has a tendency to wear down the outer side of his heel, he still uses a shoe with the sole raised on the outer side.

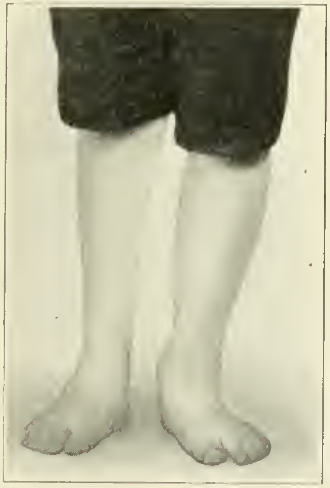

FIG. 1.—Case I. Congenital varus of the right foot, after operation.

Case II.—Tony M., aged four years, entered the service of Dr. Harte at the Orthopedic Hospital, April 26, 1906. At the age of fifteen months, some time after learning to walk, he had an attack of poliomyelitis which left his left lower extremity nearly powerless. About one year after this attack he was brought to the Orthopedic Hospital and there fitted with a brace, which he wore until admitted to Dr. Harte's service in 1906. Without the brace he could walk only with great difficulty. His foot was in the position of equinovarus, and without the brace he walked on the outer surface of his foot and on the external malleolus. There was complete loss of power in the tibialis anticus, the extensor longus digitorum, and extensor longus hallucis, in the peronei muscles, and only very slight power in the tibialis posticus, the calf muscles, and the flexor longus digitorum. The flexor longus hallucis alone appeared to be normal in its contractility, and was also a deforming factor, aiding in producing the equinus and the varus, and drawing the great toe into a position of marked flexion. The tendo Achillis was contracted, and it was impossible to bring the foot up to right angles with the leg. On May 3, 1906, under ether anesthesia, the tendo Achillis was cut, and the foot was forcibly stretched, until it could be made to assume the calcaneo-valgus position. The great toe was even more distorted by the contraction of the flexor longus hallucis, but it was not thought advisable to divide this at that time. The patient was kept in bed for twelve weeks, while the recuperative powers of the various muscles were being tried under the influence of mechanical support, massage, and electricity. At the end of this time, as very little if any improvement was noted, it was determined to try a tendon transplantation, as arthrodesis is rarely satisfactory at so early an age. The only strong tendon being the flexor longus hallucis, and this also being a deforming factor, it was decided, after consultation with Drs. Harte and Davis, to attempt its transplantation to the dorsum of the foot, through the interosseous membrane. It was thought that since the muscle lay close against the interosseous membrane, it would be more effectual to bring it directly through than to bring it around the internal surface of the tibia. Accordingly, on July 26, 1906, under ether anesthesia, and with an Esmarch bandage applied, an incision was made along the inner border of the sole, about two inches in length. The abductor hallucis was separated from the plantar fascia and the flexor brevis digitorum, exposing the internal plantar artery and nerves. These were drawn to the inner (tibial) border of the sole, along with the abductor hallucis, and the flexor longus digitorum was recognized. Between this and the metatarsal bones, the tendon of the flexor longus hallucis was detected, and its identity was proved by its function. It was then divided, and its distal end sutured to the tendon of the flexor longus digitorum, while its proximal end, after freeing the tendinous slip which passes from it to the tendon of the flexor longus digitorum above the point of section, was then drawn out of the wound. A second incision was then made along the inner side of the tendo Achillis, back of the inner malleolus, and the tendon of the flexor longus hallucis isolated just to the posterior side of the posterior tibial nerve. The tendon was then drawn out of this wound. By means of a periosteal separator the interosseous membrane was then perforated, keeping close to the fibula. A third incision was then made on the point of the periosteal separator, between the tendons of the tibialis anticus and extensor longus hallucis. The tendon of the flexor longus hallucis was then drawn through the interosseous membrane, and the point to which it would reach on the dorsum of the foot was determined. It was found to be amply long to reach to the point where the common extensor divides into its several tendons. A final incision was then made obliquely over this region, and the transplanted tendon was brought down subcutaneously to the desired point. It was here sutured, with chromic gut, to the "middle" and "ring" tendons of the extensor longus digitorum. All the incisions were then closed with chromic-gut sutures. The operation consumed nearly fifty minutes. The plaster cast was not removed until August 30, or nearly seven weeks after the operation. When the patient was told to flex his toes, the great toe and others were flexed, but the tendon of the transplanted flexor longus hallucis could be seen to contract on the dorsum of the foot. The notes say: "He walks with his ankle stiff, there being no apparent ankle-drop." As he had also paralysis of the quadriceps extensor femoris, with consequent inability to extend the knee, a brace was ordered to overcome this defect, and to support the foot until such time as further operative treatment should seem advisable. Since leaving the ward he has continued to have massage and electricity, as an out-patient, and the limb has gained considerably in strength. He can walk well without the brace, the sole of his foot coming fairly flat on the floor, whereas before the operation he could scarcely walk at all without the brace, and the outer surface of the foot and the external malleolus rested on the floor. There is still some tendency to varus, but the ankle-drop is very slight, and the power of voluntary extension of the knee has largely returned. Figs. 2 and 3, from photographs made in November last, show the location of the incisions and the extent to which the deformity has been corrected.[1]

Case III.—Eva S., aged four years, entered the service of Dr. Davis at the Orthopedic Hospital, November 18, 1902, when ten months old, with congenital equinovarus of the right foot. The foot could then readily be brought into good position She was fitted with a brace, and wore this constantly. The foot, however, could not be made to stay in the correct position without the brace. On August 29, 1905, the foot was forcibly stretched (ether) so as thoroughly to over-correct the deformity, in the hope that this would enable the weak peroneal muscles to do their part in holding the foot straight. The patient wore the brace again constantly until October 27, 1906, when tendon transplantation was done, because, though the foot was perfectly flaccid and could easily be brought into the over-corrected position, the peronei seemed absolutely powerless to hold it straight. The tibialis anticus was strong, and constantly pulled the foot into a marked varus deformity, unless

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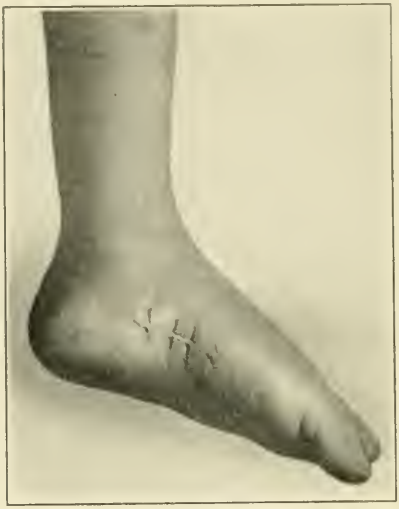
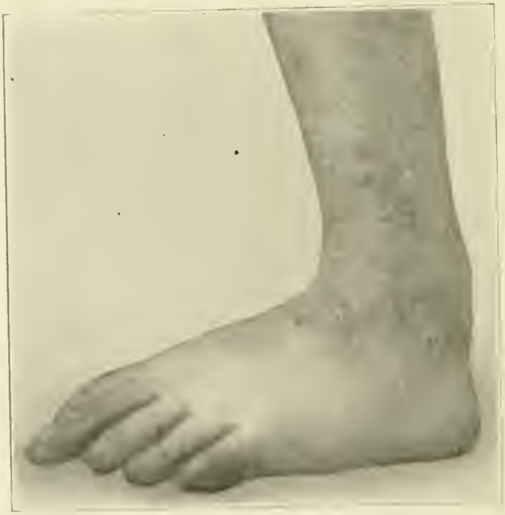
[1] Since the above was written Vignard (Lyon méd., 1907, cviii. 575, March 24) has reported transplanting the tendo Achillis through the interosseous membrane, to the dorsum of the foot, to overcome toe-drop.





FIGS. 2 and 3.—Case II. Paralytic equinovarus, after operation.

restrained by the brace. The operation in this case was precisely the same as in Case I, except that silk sutures were employed. The time was twenty minutes. After some weeks the patient was sent home still wearing the cast. While at home she developed a very severe attack of conjunctivitis, for which she was admitted to another hospital. As a consequence it was not until January 8, 1907, or nearly eleven weeks after the operation, that she returned again to the Orthopedic Hospital. Removal of the cast on that date showed that the skin wound over the outer side of the foot had not entirely united, and that two silk sutures were protruding. These were removed, and a cast was again applied. January 15, this cast was removed and the skin wound was found to have united solidly. She was directed to wear an ordinary shoe and return in four weeks. On February 12, it was found that active motion pulled the foot into varus, suggesting that the tibialis anticus had slipped from the tuberosity of the fifth matatarsal bone, when the silk sutures separated by sloughing. On March 1, the patient was etherized, and the tibialis anticus separated from the dorsum of the foot, where it had become adherent between the first and second metatarsal bones, and again sutured to the tuberosity of the fifth metatarsal bone, with silk sutures. The result this time was excellent.

Case IV.—Mary M., aged twenty years, entered the service of Dr. Davis at the Orthopedic Hospital, March 23, 1897, when twelve years of age. She had had an attack of poliomyelitis when about one year old. She was wearing a brace which had been ordered elsewhere. Without the brace her foot assumed a fairly marked varus deformity, owing to the paralysis which affected chiefly the peroneal muscles. The patient neglected treatment, returning once to the hospital in May 1898, but not again until November 20, 1906. It was now found that a moderate degree of varus persisted, owing to paralysis of the peronei, and that the tibialis anticus was also very weak. Dr. Boyer, who examined the foot electrically, reported that the tibialis anticus and peronei responded slightly to both galvanic and faradic currents, but not normally. No reactions of degeneration were present, however. The patient was very stout, and her foot was constantly turning under her; indeed, she stated that for some months she had been unable to walk a square without falling. Fig. 4 shows the degree of deformity present, as well as the contraction of the great toe due to the action of the extensor longus hallucis. On December 29, 1906, the patient was etherized, and the extensor longus hallucis was divided on the dorsum of the foot, its distal end being sutured to the neighboring tendons of the extensor longus digitorum, and its proximal end being transplanted to the tuberosity of the fifth metatarsal bone. It was sutured to the periosteum at this point by four chromic gut sutures. The patient had been operated on by tracheotomy when a child, and she took ether badly; the operation was also done without an Esmarch bandage. For these reasons the time consumed was an hour. The cast was changed January 29, 1907, but was not finally removed until February 26, when the patient was directed to wear an ordinary shoe. Fig. 5 shows the foot in the position obtained by the operation and the absence of contraction of the great toe. Flexion of the ankle and eversion of the foot are well performed by the transplanted tendon.

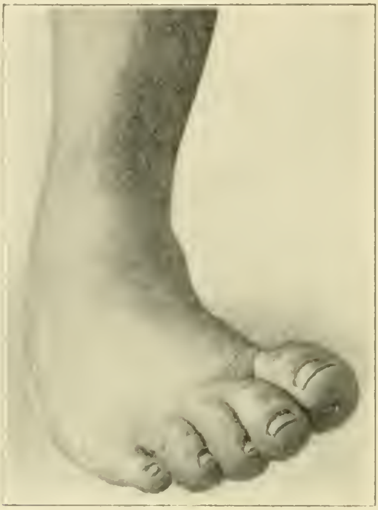


FIG. 4.—Case IV. Paralytic varus of the right foot, before operation.

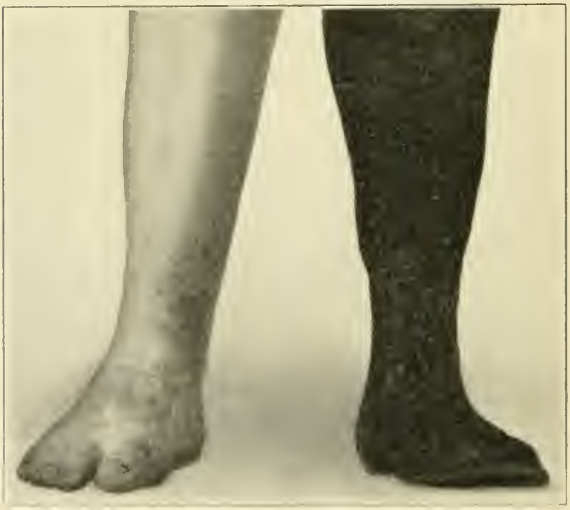


FIG. 5.—Case IV. Paralytic varus of the right foot, after operation.

CASE V.—Leslie E., aged fifteen years, entered Dr. Harte's service at the Orthopedic Hospital in November, 1906. At the age of seven years he suffered from an acute febrile attack, which left him with partial paralysis of the right leg. Ever since, that is, for eight years, he has worn a brace. There is marked varus and cavus, with slight equinus. There is outward luxation of the head of the astragalus. There is atrophy of the calf, although some power of extension of the foot persists, but there is complete paralysis of the peronei. Without the support of the brace, he stands on the dorsum of the foot (Fig. 6). On November 11, the patient was etherized, the plantar fascia was cut, the arch was depressed with the lever, the varus was partly corrected by the varus machine, and the tendo Achillis was finally divided. The foot then came into the over-

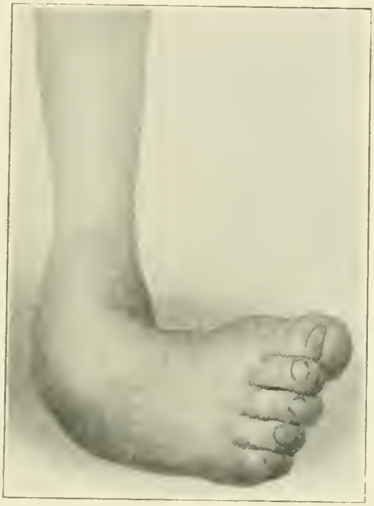


FIG. 6.—Case V. Paralytic varus of the right foot, before operation.

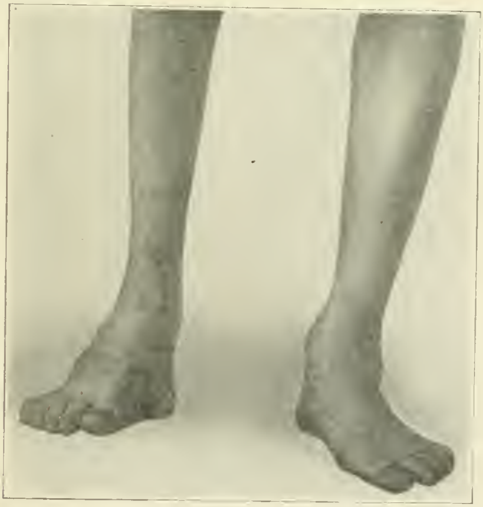


FIG. 7.—Case V. Paralytic varus of the right foot, after operation.

corrected position only with great tension. Put up in plaster. The cast was removed November 29, and after receiving massage for a week or ten days, the patient was again etherized on December 14, and the foot was again stretched, very forcibly, with the lever, and over the triangular block. The projecting astragalus was thus mashed into place, and the foot assumed quite a normal appearance. The cast was left on until January 2. On January 16, 1907, the patient was etherized for the third time, and the tendon of the tibialis anticus was transplanted to the tuberosity of the fifth metatarsal bone. Owing to the greater size of the foot it was not deemed advisable to attempt subcutaneous division of the annular ligament as in Cases I and III, so an incision was made over the tibialis anticus above the annular ligament, the tendon was here drawn up (after separating it from its insertion through the first incision), and was thence drawn through a subcutaneous channel to the desired point of suture over the tuberosity of the fifth metatarsal bone. The tendon was sutured to the periosteum in this place by four chromic gut sutures. The time consumed was thirty minutes. The plaster cast was left on until February 9, when it was changed. The patient was now allowed to walk about the ward with his cast for a shoe. The cast was finally removed on February 28, more than six weeks after the operation. The transplanted tendon contracted firmly in its new position, and produced good flexion at the ankle, with eversion of the foot (Fig. 7). With a shoe having the outer side of the sole raised a quarter of an inch it is difficult to detect in the gait any limp except that due to the general atrophy of the right lower extremity, which is an inch shorter than the left.

Case VI.—Lizzie McQ., aged seven years, entered Dr. Davis's service at the Orthopedic Hospital, January 1, 1907. She had had an attack of poliomyelitis before she learned to walk. There was complete paralysis present of the tibialis anticus; and the peronei, the extensor longus digitorum, and the extensor longus hallucis all produced a fairly marked condition of valgus (Fig. 8). It was determined to transplant the peroneus brevis and the peroneus tertius into the tibialis anticus at its insertion. On January 25, 1907, an incision was made over the insertion of the peroneus brevis, and this was detached from the bone. An incision was then made above the external malleolus, behind the fibula, and the tendon of the peroneus brevis was drawn up. A third incision was made over the insertion of the tibialis anticus, and the tendon to be transplanted was drawn down subcutaneously to this point, where it was sutured with four silk sutures to the tibialis anticus (the foot being held in the calcaneovarus position), and with two other silk sutures to the periosteum of the scaphoid bone. Then an incision was made over the peroneus tertius, which was detached from its insertion and transferred subcutaneously across the dorsum of the foot to the inner side, where it was sutured to the tendon of the tibialis anticus and to the periosteum with three silk sutures. The incisions in the skin were all closed with chromic catgut. The time of the operation was twenty minutes. A plaster cast was applied, and not removed until March 26, more than eight weeks after the operation. The transplanted tendons were found firmly united to the tibialis anticus. By their contraction the arch of the foot is restored (Fig. 9), and the foot instead of being pulled by flexion into the extreme valgus position as was the case before the operation, is now flexed without appreciable deviation from the mid-line. The patient wears an ordinary shoe, and does not limp.

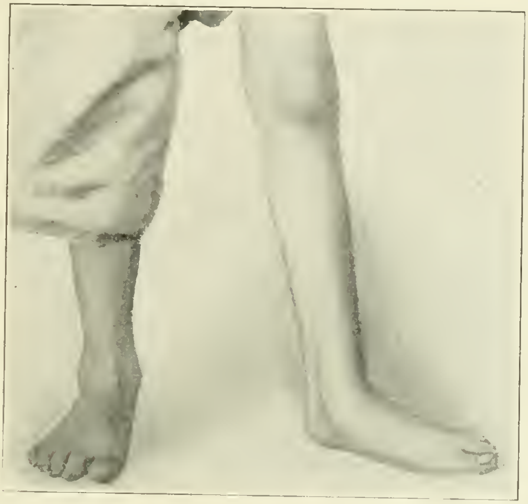


Fig. 8.—Case VI. Paralytic valgus of the left foot, before operation.

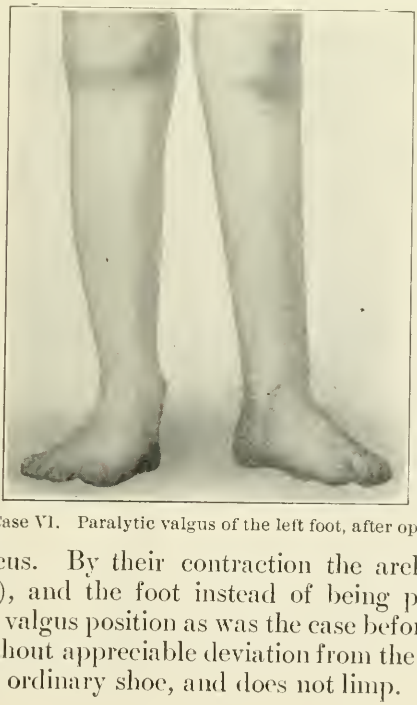


Fig. 9.—Case VI. Paralytic valgus of the left foot, after operation.

FURTHER REMARKS ON THE CARE OF GUNSHOT WOUNDS OF THE ABDOMEN.

WITH ESPECIAL REFERENCE TO THE TREATMENT OF WOUNDS OF THE URINARY BLADDER AND THE DANGER OF ABSCESS AND FISTULA FOLLOWING WOUNDS OF THE PANCREAS.

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Last year I[1] reported 14 cases of gunshot wound of the abdomen. Since that time I have had 3 more cases, which I desire to report with comments, as 2 of them were of unusual interest.

Case I.[2]—*Ten holes in the small intestine, two in the bladder; resection; permanent catheterization; recovery.*

W. H., a colored male, aged twenty-three years, a native of Maryland, a waiter by occupation, was operated on January 27, 1906, at the Emergency Hospital, four hours after having been shot. A 0.32 pistol-ball had entered the abdomen on the right side, one inch to the inner side of the anterior superior spinous process of the ilium. The condition of the patient was good; his pulse 96; he had vomited once, and had some pain in the bowels, but it was not very severe. Under ether anesthetization the abdomen was opened through the right rectus muscle, revealing considerable thin, bloody fluid, ten holes in the small intestine (ileum) and two in the bladder. The ten holes in the bowel were in a portion of bowel not over two feet in length, and four of the holes were in a section not over two inches long. Six holes were closed with silk sutures; the two inches of bowel so badly lacerated were resected and an end-to-end union made with silk sutures. One opening in the bladder was closed, but the other was so deep in the pelvis as to be inaccessible without opening the bladder and suturing it from the inside. Rather than do this it was decided to provide permanent drainage to the bladder, in order to prevent the escape of urine into the peritoneal cavity; this was done by putting into the bladder, through the urethra, a soft-rubber catheter and fixing it in position. The abdominal incision was closed, leaving a rubber drainage tube in the lower end of the incision. There was suppuration in the abdomen for about three weeks, when it ceased and the drainage tube was removed. The catheter was removed twice a week for cleansing and was left out permanently at the end of three weeks. The patient had no difficulty in urinating. He was permitted to get up March 1, thirty-two days after the operation, and was discharged recovered March 12.

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[1] Amer. Jour. Med. Sci., February, 1906.

[2] Case XV, continuing the former series.

A skiagram showed the bullet on the left side, apparently just above the neck of the left femur. It was not disturbed.

CASE II.—*Nine openings in the small intestine, two in the bladder; operation twelve hours after injury; death.*

R. W., colored, male, aged twenty-eight years, was shot March 1, 1906, the ball entering the back near the spine, just below the left twelfth rib, and was operated on at Georgetown University Hospital March 2, about twelve hours after the injury. Pain did not come on for several hours after the shooting, but when seen twelve hours later the patient was in great pain. The abdomen was swollen and tender; he had passed bloody urine, had vomited once, and the pulse was 110. Under ether anesthetization the abdomen was opened in the median line, just above the pubes. At least three pints (1500 c.c.) of bloody fluid with a fecal odor was found in the peritoneal cavity, and peritonitis was well advanced, the intestines being distended, swollen, and many coils covered with lymph. Nine holes were found in the bowel, distributed through about the last seven feet of the ileum, and two in the bladder, one above the other, about one and one-half inches apart. All the holes were closed with silk sutures, the abdominal cavity was thoroughly irrigated with salt solution and closed with drainage, and a catheter was fastened in the urethra. The ball, 0.32 in calibre, was found loose in the bottom of the peritoneal cavity. Death from peritonitis occurred twenty-six hours later.

CASE III.—*Wounds of the liver, stomach, pancreas, and kidney; primary healing after operation, followed two months later by a second operation for abscess in connection with the pancreas.*

Mrs. A. K., a white female, aged twenty-five years, was shot June 25, 1906, and was operated on one and one-quarter hours later at the Emergency Hospital. A 0.32 pistol-ball had entered the epigastric region three inches below the ensiform cartilage and one inch to the left of the median line; it could be felt beneath the skin on the back, just below the last rib and three inches to the left of the median line. At the time of the operation the patient was in good condition, pain was not severe, the pulse was 100, the skin cool, and she had vomited blood. Under ether anesthetization the abdomen was opened through the left rectus muscle. A hole was found in the left lobe of the liver from which bleeding was still going on, and one in the anterior wall of the stomach. These were sutured at once, the liver with catgut and the stomach with silk. The lesser peritoneal cavity was then opened by bringing up the transverse colon and splitting the mesocolon, thus exposing the posterior wall of the stomach, in which another hole was found and sutured. A hole in the pancreas was sutured; beyond this the ball was not traced, but there can be no doubt that it wounded the left kidney, as the organ lay directly in the path of the bullet, and examination of the urine showed considerable blood which could not otherwise be accounted for. No irrigation was used in the toilet of the peritoneum; the blood and clots were simply sponged out and the abdomen was closed without drainage. There was no trouble about healing, but the temperature and pulse continued above the normal. On July 27 the ball was removed under cocaine, but no sign of abscess was seen.

On August 25, two months after the operation, the patient was still not well, and during the last two or three weeks she had been getting worse. The temperature had not remained normal for more than a day at any time since the operation, usually ranging from 99° to 101°, the pulse 90 to 110, although she had been sitting up and walking about. Recently the temperature had been running higher, 100° to 103°, the pulse 100 to 120; there were no chills, but sweats were common, appetite was poor, constipation was present, and marked loss of flesh had occurred. Physical examination showed tenderness, swelling, and abnormal percussion note. It was thought that an abscess might be present, possibly due to the action of the pancreatic juice, and an exploratory laparotomy was done August 25. The abdomen was opened through the scar. Spider-web adhesions were found, but the wounds in the liver and stomach (anterior) were well healed. On lifting up the transverse colon a hard mass was felt deep in the cavity, behind the lower border of the stomach, bounded above by the pancreas, with which it was attached; to the outer side was the descending colon; to the inner side the inferior mesenteric vein showed prominently, while also to the inner side, below and in front, was the first portion of the jejunum, just after the intestine escapes from the ligament of Treitz. These parts were adherent, and on breaking up the adhesions a cavity containing about a tablespoonful of yellowish debris was discovered. The stomach did not appear to enter into the formation of the abscess. The broken-down material was wiped out and a rubber drainage tube was put in through a stab wound through to the back, where it was sewed to the skin. The incision in front was closed without drainage. Four days later the pulse and temperature were practically normal and remained so. There was only a slight discharge through the tube until the fourth day, when a large quantity of what looked like pus, and contained staphylococci, was discharged. The discharge continued for five weeks, when it ceased and the opening healed. It was collected in two or three days and amounted to seven ounces in twenty-four hours, and on being tested by Dr. J. F. Anderson, it gave all the reactions of pancreatic juice.

The first 2 cases show quite clearly the importance of early operation. The wounds were almost exactly alike in location and severity, but the one patient was operated on four hours, and the other twelve hours, after the reception of the wound; there was no evidence of peritonitis at the time of operation in the first case, but peritonitis was well under way in the second case and death resulted.

Another point of interest is the use of the catheter as a drainage tube for the bladder, to prevent the escape of urine into the peritoneal cavity when, for any reason, a wound of the bladder cannot be closed. I generally use it even in wounds of the bladder which have been completely closed, in order to prevent tension while the wounds are healing.

Case III is interesting on account of the pancreatic fistula and its resemblance to the wounds received by President McKinley, in which the bullet passed through the stomach and wounded the pancreas and left kidney, and was followed by the formation of an abscess in connection with the pancreas.

THE CAUSES AND TREATMENT OF STERILITY IN WOMEN.

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The treatment of sterility is mainly minor office treatment and has long been regarded as one of the most unsatisfactory parts of gynecological practice. That positive results cannot be promised is still true; unsuccessful cases are still met with, and always will be; yet, in point of fact, it is not nearly so discouraging as it has been considered. The subject usually has not been studied in sufficient detail, and the treatment of a given case is too frequently based on an insufficient study of the patient.

Of course, it must be premised at the outset that the fertility of a given marriage is dependent upon the condition of both of the two partners. There has long been a general impression that sterility is exceedingly rare in men and common in women, an erroneous conception which has led, and still leads, to much unnecessary and unfair treatment of perfectly fertile women; but it is as a result of this conception that it is usually the woman who presents herself for treatment.

It is probably true that sterility is somewhat more common in women than in men, and when the woman who comes for treatment presents some minor abnormality which might prevent conception, and which can easily be remedied by painless treatment, it is probably wise to apply such treatment for a reasonable length of time before questioning the condition of the husband; but no extended or uncomfortable treatment should be given, and certainly no operative work, even of a minor nature, should ever be undertaken without first establishing the fertility of the husband. Sterility in men is not, however, the subject of this paper and may, therefore, be dismissed with this preliminary proviso.

Sterility in women may be dependent on many possible causes, the potential existence of each of which must be carefully weighed in each case, and, as will be seen, each case involves for successful treatment a careful clinical study of the whole genital system of the patient as a preliminary undertaking before any treatment is instituted.

One fact of the utmost importance, however, has been so generally overlooked as to require emphatic consideration at the very beginning of any study of this subject. Sterility in women may be dependent, as has been said, on many and very diverse causes, but, whatever the original cause may be, the immediate factor by which conception is prevented is, in the vast majority of remediable cases, an alteration of the secretions of one or another part of the genital tract of such a nature as to render them hostile to the ovum or the spermatozoa. We have then two points which are always of diagnostic importance. The clinical key to the diagnosis and treatment of any given case lies in the study of the secretions, but on the other hand it must not be forgotten that the determination of the lesion or condition which underlies any change of secretion is essential to its rectification, hence the necessity for the most careful inquiry into the condition of the entire genital canal at the beginning of every case.

The causes of sterility may be conveniently classified for analysis from a clinical standpoint as follows:

NOT EASILY REMEDIABLE. (*A*) Imperfect ovarian function; that is, ovaries which fail to produce a perfect ovum, capable of impregnation.

(*B*) Organic or functional imperfections of the tubes; that is, tubes which are incapable of transmitting the ovum to the uterus.

REMEDIABLE. (*C*) An inhospitable endometrium.

(*D*) Hostile secretions.

The subject of the treatment of the not easily remediable causes of sterility need be given but briefly here, since it has of late occupied much space in gynecological literature in the course of the discussion of the conservative surgery of the appendages. Certain general principles, however, must be summarized before we pass to a fuller discussion of the less familiar and much more easily remediable minor causes of sterility.

(*A*) OVARIAN IMPERFECTION. The existence of ovarian imperfection is usually clinically uncertain, whether in small, normal-sized, or enlarged ovaries, and is capable of positive proof in a given case only when the ovaries are exceedingly small, either from senility or from arrested development. Underdevelopment of the ovaries probably always implies underdevelopment of the uterus also, and its prognosis depends on its degree.

If imperfect function coexists with a normal-sized ovary it cannot be diagnosticated. The existence of a merely functional incapacity has not been definitely demonstrated, but that it does occasionally appear is inherently probable, and this probably explains some of the otherwise unexplained failures of fertility in apparently normal women. It is a practical point that the possibility of this cause must always render the prognosis of a given case doubtful, especially in nulliparæ, but the more carefully our cases are studied the less frequently are we obliged to fall back upon this possibility.

Enlargement of the ovaries to a moderate degree is not necessarily a cause of sterility even though it be bilateral, as is shown by the many cases in which pregnancy has been observed to occur with somewhat enlarged ovaries and even in the presence of large ovarian neoplasms; that cysts of one or both ovaries may inhibit pregnancy has been conclusively shown, on the other hand, by the many reported cases in which the conservative surgery of moderately enlargd ovaries has been followed by conception in previously sterile women. In spite, however, of the large aggregate number of isolated successes which have now been reported, a wider study of the records of all conservative operations on the ovaries will convince anyone that the fertility of such women is well below par even after operation, that is, no large percentage of them ever conceive; and though want of space forbids an extended discussion of this subject, it may probably be laid down as a rule that the conservative surgery of the ovaries does not as yet offer a sufficiently good prospect of fertility often to warrant the opening of the abdomen for the treatment of small enlargements of the ovary on account of sterility alone, and in the absence of other symptoms. In other words, the value of such surgery lies rather in the possible preservation of fertility in women whose general ill health warrants operation, than in any hope of producing fertility in sterile women who are otherwise in good health.

(*B*) Imperfect Action of the Tubes. This may be dependent upon (1) organic stricture and (2) alterations of the mucous membrane.

1. *Organic strictures* are probably always inflammatory in their origin, and hence usually involve a coincident abnormality of the mucous membrane. They can be diagnosticated as present only when the existence of distention or adhesions is sufficiently marked to be palpable. Their treatment, of course, is purely operative, and the prognosis must depend upon the situation of the closure and the condition of the mucous membrane.

2. *Alterations of the mucous membrane* of the tubes are probably always inflammatory in one degree or another. That exceptionally they may be caused by disturbances of menstruation independent of infection is undoubted, but they are far more often the product of infection, obstetric or gonococcic. Their prognosis is always doubtful, but fertility is rare after an infection of the tubes has produced a palpable lesion. Their treatment apart from the operative management of closed tubes is wholly that of the management

of the acute stages of the inflammatory diseases of the tubes, and that of the endometrium, to be spoken of later.

(*C*) The Inhospitable Endometrium. This subject, in its relation to sterility, resolves itself clinically into two divisons: (1) The results of infection, and (2) hyperplasia from chronic congestion without infection.

1. *Infection of the endometrium* almost always involves infection of the mucous membrane of the tubes, and therefore needs no further discussion here except to say that even in the comparatively rare cases in which spontaneous recovery from infection is so complete as to involve a possible restoration of function, restoration of fertility is usually inhibited by a hyperplasia due to the long-continued congestion which is a concomitant of the endometritis,[1] and the treatment is then that of hyperplasia from congestion.

2. *Hyperplasia from Congestion.* The chronic congestion which underlies this condition is probably always due either to some other abnormality in the genital tract or to constitutional conditions. Its successful treatment, therefore, is dependent upon a detection of the underlying causes of the congestion and upon their removal before any treatment of the hyperplasia as such is undertaken. The mere enumeration of all the causes of congestion of the uterus would manifestly open too wide a subject to be entered upon here, but it may be said that after the removal of the cause the hyperplasia itself will often persist and demand curettage.

The diagnosis of the minor conditions of the endometrium which render it hostile to the ovum or spermatozoa, and which, therefore, are causes of sterility, is, however, a subject of much practical importance, which has been much neglected, and which is essentially pertinent to the present paper.

From the standpoint of sterility this, which I have called the hostile endometrium, is then usually to be detected clinically by observation of the uterine secretion. In almost all of the easily remediable cases the actual bar to conception is found in an alteration of the uterine or vaginal secretions, or both, of such a nature as to be detrimental to the spermatozoa. The most hopeful and essential part of the treatment of sterility is to be found in the study of the abnormal conditions of these secretions, and their restoration to the normal, both by the removal of the causes of their alteration and also by direct treatment of the secreting surfaces themselves.

(*D*) Hostile Secretions. Of necessity a knowledge of the hostile secretions presupposes a knowledge of their normal state, and of the conditions necessary to their functional efficiency, for the comprehension of which some knowledge of what their physiological functions are is in itself a necessity.

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[1] The classification of the inflammatory conditions of the endometrium has of late become so confused by conflicting pathological views that I prefer not to enter into this subject in a clinical article even by implication, and have therefore limited myself here to the rather non-committal terms of endometritis and hyperplasia.

1. *Uterine Secretion.* All mucous membranes are moist and covered with secretions of varying characteristics. It is probable that the function of the uterine secretion is simply that of affording a medium through which the spermatozoa may pass to the ovum, and for this purpose its condition should be, as in the normal state it in fact is, that of a mildly alkaline albuminous fluid of very limpid consistency, somewhat scanty in quantity (insufficient for a perceptible flow from the os), clear, and so slightly viscid as to form a rounded drop without stringiness when withdrawn from the cervix.[2]

The secretion of a congested or simply hyperplastic endometrium is usually clear, alkaline, profuse, and somewhat increased in consistency over the normal; that of an endometrium which has been infected varies from cloudy to purulent and from viscid to semisolid; it is usually alkaline, but may be faintly acid. The normal alkalinity of the uterine secretion may be decreased even to neutrality by the constitutional conditions which lead to an overacidity of all the secretions, but an actual acidity of this secretion is rare.

2. *Vaginal Secretion.* The functions of the vaginal secretion are essentially different and, indeed, in part opposed to that of the uterine secretion. Its presence is not necessary or even favorable to the spermatozoa, which are normally deposited surrounded by their own fluid in contact with the os. It subserves the two functions of lubricating the vaginal walls, which are always more or less movable upon each other during the movements of respiration, defecation, etc., and of affording an obstacle against the infection of the mucous membranes of the uterus and tubes (and therefore of the peritoneum) by the introduction of bacteria from without. This function is of the first importance, both to the health of the individual and to the preservation of the race, and its bearing on our subject makes it worthy of some little exposition here.

The bactericidal properties known to be inherent in the normal vaginal secretion are probably twofold, consisting, first, in its normally acid reaction, and, second, probably, in the normal existence within it of bacteria whose activities are antagonistic to the existence, or at least to the virulence, of the pathogenic forms. The normal vaginal secretion contains two somewhat different components. It has been described by Döderlein and Williams as consisting of a small quantity of white material not unlike curdled milk or partly liquefied cream cheese, scanty in amount, and very acid in reaction; but these observers have evidently neglected, or neglected to state, the fact, patent to anyone who will look at the normal vagina, that the greater part of its secretion consists of a thin, clear, watery fluid which is very moderately acid, very slightly slippery in consistency, and which should be present in sufficient amount to keep the vaginal walls moist, but not in amount sufficient to exude as fluid from the vulva. It is normally sufficiently scanty to dry from the labia as soon as it reaches the external air; scattered through this are small masses of the white, curdy material described by Döderlein, and accepted by Williams and others as the normal secretion of the vagina. This white material is probably always present, but is very scanty in the secretions of virgins, is always more abundant in patent vaginae, and is present in profuse quantity in the secretions of pregnant women. As the observations of Döderlein and Williams, like those of most workers on the bacteriology of the vagina, were undertaken with especial reference to puerperal infections, and conducted for the most part on the secretions of pregnant women, the reason for their especial emphasis of the presence of this white material is sufficiently obvious, but it is in fact the less constant of the two elements of the secretion. It may possibly be the product of Bacillus lactiformans of Döderlein.

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[2] Although some action of suction has been attributed to the uterus during coitus by numerous theorists, this somewhat fanciful theory is not founded upon any scientific basis, and is decidedly opposed to the little that we know of the process of impregnation in the lower animals. It is probable that the only method by which the spermatoza can reach the cornua is by their own natatory efforts, for which the existence of the uterine secretion is essentially necessary.

In spite of the extensive observations which have been made by Döderlein, König, Williams, and many others upon the bacteria of the vagina, our knowledge of its bacteriology is still but rudimentary, the whole subject being obscured by the habitual presence in it of a great variety of non-pathogenic species, in addition to which some of the observers have reported the presence of forms which would be considered pathogenic if they had not apparently lost all virulence. The only assured knowledge which we have is that which has been stated above, that it is bactericidal, and that this property is in part due to the presence and activity of non-pathogenic species, in addition to the inhibition offered by its acid reaction. This latter is probably the more important.

It is well known, to quote from Neumann, that "Most varieties [of bacteria] of practical importance, and all pathogenic varieties, prefer a medium containing albumin which is faintly alkaline in reaction." In connection with the acid-producing capacities of many bacteria a great amount of experimental study has been made upon the capacity of all the important species to withstand acid media, but since the degrees of acidity at which they reproduce themselves even sparsely are too slight to be easily detected by litmus paper and are consequently calculated by the quantity of a 1 per cent. alkaline solution necessary to neutralize a similarly prepared acid medium, while the normal vaginal mucous membrane stains blue litmus paper a moderate red, that is, shows it to be of a degree of acidity which makes an unpropitious if not impossible culture medium, it is evident that this acidity is in itself a bactericidal factor of the utmost importance; indeed, this has been admitted by all observers.

That the vaginal secretion possesses, however, a further bactericidal action has been established by repeated experiments in which the pathogenic varieties failed to grow, or grew but sparsely and with loss of virulence, when carefully neutralized vaginal secretion was employed as the medium. Hewlett sums up the situation by saying, "There [in the vagina] we have a normal flora which may injure strange invaders, and the acid reaction is unfavorable to most pathogenic bacteria."

That the natural bars against infection, however, are not wholly efficient is shown by the facts that the gonococcus does not lose its vitality in moderately acid media, and that if other bacteria are introduced in sufficient quantity, or surrounded by enough alkaline fluid, they may retain their virulence long enough to reach the alkaline media of the uterine secretion. It would have seemed easy for evolutionary processes to have protected the all-important reproductive functions against infection more surely by the interposition between them and the external world of a more strongly acid, and therefore more bactericidal, vaginal secretion, were it not for the fact that the spermatozoa, like other protozoa, are incapable of survival in any but a neutral or faintly alkaline medium. It is therefore necessary to the function of reproduction that the vaginal secretion should be so moderate both in quantity and acidity as to be easily displaced or neutralized by the alkaline semen which surrounds the spermatozoa (it is well known that the use of a slightly acid injection immediately before coitus is one of the most successful methods of preventing conception), hence the evolutionary compromise that a vaginal secretion which shall act as a bar to infection[3] without preventing conception must be scanty in quantity and faintly acid; and, in point of fact, the existence of a profuse and improperly acid vaginal secretion is one of the most frequent and efficient causes of sterility. The acidity of the vagina is due to the presence of lactic acid, which is believed to result from the activity of Döderlein's (lactic acid producing) bacillus, a fact which is essential to our understanding of some of the factors in the treatment of the abnormal secretions for the relief of sterility.

The vaginal secretion may be altered from the normal in quantity, reaction, consistency, clearness, and color. Its variations in chemical reaction may be of constitutional origin or due to bacterial activity; it may be greatly increased in quantity without other changes by simple congestion; but all changes other than these two are probably due to the activities of bacteria, and are therefore the result either of an increase in the normal flora or of the introduction of pathogenic species.

CHANGES IN THE SECRETIONS FROM MERELY CONSTITUTIONAL CONDITIONS. Acidity of the uterine secretion or the more common excessive acidity of the vaginal secretion is one of the most important conditions from the standpoint of the relief of sterility, since, unimportant as it otherwise is, it is during its persistence necessarily prohibitive of fertility. It is the only change in the secretion which can be due to merely constitutional conditions, that is, which may be independent of local causes. It may be produced by any state which tends to acidity of all the secretions, and when the acidity of both secretions is increased without other changes the cause is always constitutional, but it must be remembered that even when it is in part of fermentative origin there may also be a constitutional element present, so that this must always be looked for. What that element may be is to be determined by a quantitative examination of the twenty-four-hour urine, which in such cases will always show an excess in the production of one or the other of the acid elements, and will thus indicate the dietetic changes which should be employed to produce a decreased acidity of all the secretions.[4]

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[3] The vagina, like all external canals, is constantly exposed to infection from without.

CHANGES DUE TO BACTERIAL ACTIVITY. In addition to the always important changes in chemical reaction we must study the changes in the quantity, consistency, and color of both secretions, but more especially that of the vagina. The many observers who have worked upon the bacteriology of the vagina have usually confined themselves to the changes in the secretion which are produced by the presence of the pathogenic bacteria, and have therefore described only the normal secretion and the secretions found in cases in which the pathogenic species were found to be present, but it must be remembered that their work was founded mainly upon the puerperal discharges and was conducted throughout with especial reference to the etiology of puerperal infection.

From the standpoint of the study of fertility we must recognize clinically three states of the vaginal secretion: (1) The normal. A clear, serous secretion, moderate in quantity, faintly acid in reaction, and containing varying quantities of white, curdy material of very acid reaction. (2) A somewhat more profuse discharge, not distinctly purulent, but turbid, yellow in color, of greatly increased acidity, and usually, though not always, free from the presence of the normal cheesy material. (3) An abundant creamy discharge which may be purulent, seropurulent, or mucopurulent, and is usually in addition somewhat excessively acid in reaction.[5] This last form of discharge is always significant of the presence, or past presence of the pathogenic species, as has been established by numerous bacteriological observations, and usually affects both secretions. The other form is usually limited to the vagina and is probably significant of an overactivity of its normal flora, but this point has not yet been studied bacteriologically. This is a point well worthy of such observation, and I hope to be able to effect its accomplishment in the future.

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[4] Excess in the use of common salt is a not uncommon cause of constitutional overacidity of the secretions.

[5] During the acute stages of a pathogenic infection the secretion is alkaline, but usually becomes acid as the virulence of the invasion passes off.

To sum up, then, the changes which should be looked for in the secretions are:

(*a*) *Change in Both Secretions Simultaneously.* We may find both secretions changed together in the direction of overacidity, but without other changes; this is always constitutional overacidity. We may have both secretions changed together in the direction of increased quantity, but without other changes; this is the result of simple congestion. We may have both secretions changed together into a seropurulent, mucopurulent, or purulent discharge; this is always the result of an infection by pathogenic bacteria.

(*b*) *Uterine Secretion Normal, Vaginal Secretion Abnormal.* Since the uterine secretion is discharged into the vagina we never see the uterine discharge affected while the vaginal discharge is unchanged, but the vaginal may be abnormal without any change in the uterine secretion. This is either temporary, that is, the initial stage of an ascending pathogenic infection, or more or less permanent as a result of non-pathogenic bacterial action, that is, an excessive acid fermentation.[6]

METHODS OF CLINICAL DIAGNOSIS OF THE SECRETIONS. The vagina should be opened by a speculum before any other form of an examination is made, and without the use of a lubricant in introducing the speculum.[7] The quantity, color, and consistency of the vaginal secretion is now noted and its reaction taken. The vaginal walls and the vaginal surface of the cervix are then carefully cleansed with dry cotton, no solution being used for fear of changing the reaction. The appearance of the cervical secretion is then noted either by spreading the lips apart by tension on the vaginal walls with the speculum or by compression of the cervical canal between the blades of the speculum or between the anterior blade of the speculum and a pledget of cotton pressed by forceps against the posterior aspect of the cervix. The drop of cervical secretion so brought to the os is then tested with litmus paper (the litmus may if necessary be passed into the cervical canal by the forceps, but if this is done care must be taken not to excite bleeding, which will at once ensure a stronger alkaline reaction); the color of the vaginal and the cervical mucous membranes is noted and a careful bimanual examination of the uterus, tubes, and ovaries is then made. The constitutional condition of the patient is canvassed and a twenty-four-hour specimen of urine is collected and sent for quantitative analysis. Not until all this has been done is an observer in a position to determine the cause of a sterility.[8]

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[6] The etiology of this change has not been definitely proved, but it is a fair working hypothesis that it is due to the presence of excessive quantities or unusual varieties of the non-pathogenic bacteria, *i. e.*, that it is an excessive acid fermentation of the vaginal discharges. It is certain, from clinical observation, that a yellowish and intensely acid vaginal secretion may be persistent for a long time without effecting any change in the uterine secretion.

[7] The introduction without a lubricant is rendered easy and painless to the patient if the labia are first drawn apart by an assistant so that the speculum does not come in contact with the dry external skin, but passes at once into the moist and slippery vagina. A small sized speculum should, of course, be used.

CLINICAL TREATMENT OF THE HOSTILE SECRETIONS. The treatment of overacidity of constitutional origin is, of course, that of the constitutional condition present; its full discussion would occupy too much space to be entered upon here. It may suffice to say that after the determination of the elements of acidity by an examination of the urine, the appropriate diet and hygiene should be selected experimentally, and tested and modified by repeated quantitative analyses of the urine. It is not infrequently connected with the excessive ingestion of salt, as evidenced by an excess of chlorides in the urine.

The treatment of excessive quantity of the secretions due to congestion is, of course, that which is likely to remedy congestion. When sterility is due to this cause it will occasionally happen that a mere depletion of the pelvic organs by glycerin, preferably in the form of suppositories inserted each night for some weeks, will result in conception, but more often it is necessary to remove permanently the congestion by the detection and treatment of the lesion or condition which causes it.

When a congestion is dependent upon a retroversion, new-growth, or other gross lesion of the organs, the accompanying sterility is usually only one of the symptoms for which the patient seeks treatment. An elaborate discussion of such cases would manifestly cover almost the whole field of gynecological practice; but the existence of congestion without apparent lesion is usually indicative of an abnormality in the functional use of the organs. Sexual perversion I believe to be extremely rare, at all events among the women whom I have been called upon to treat. In a somewhat extended experience I have seen but two instances of it, and both of them were cases of psychical rather than practical perversion. Self-abuse I believe to be rare among women, but numerous abnormalities of desire, such for instance as the capacity for excitation with but little if any power of relief by obtaining the orgasm, I know to be extremely common and a most fertile cause of congestion of the pelvic organs. The results of these abnormalities of function in the production of neurasthenia, pelvic pain of the neurasthenic type, etc., form a subject of much interest and importance which for obvious reasons has been seldom or never written upon, and into which it is not my present purpose to enter. I must content myself here with expressing my belief that he who would successfully treat sterility must be equipped with some knowledge of this subject, and in spite of its disagreeable features should not hesitate to enter upon it if he would do justice to his patient, whenever he believes that the sterility for which he is consulted is the result of an otherwise unexplained congestion; this is the more especially true if the patient has acquired or increased a neurasthenia since marriage.

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[8] The conditions under which the fertility of the husband should also be investigated have been already referred to.

A purulent or semipurulent condition of both secretions is always the product of a pathogenic infection. Neither the acute or subacute stages of such an invasion comes properly within the province of a paper on sterility, since during those stages the patients consult us for symptoms of the acute infection, and it is only after their subsidence that the question of the resultant sterility comes up clinically. The majority of such infections leave behind them one of the lesions of the tubes or of the endosalpingium which have been referred to above, and this point must always be considered in the diagnosis of the chronic cases; it is only those which progress more favorably which concern us here.

During the acute stage the secretions are usually actively purulent; during the resolving stages, which cover weeks, months, or more often years, the secretion which issues from the os changes gradually through a seropurulent to a mucopurulent condition, and the attack resolves itself finally to a condition in which the secretion is composed of a very evident semisolid, stringy mucus, around which a thinner, viscous, and more abundant cloudy fluid exudes. In most such cases the semisolid mucous plug is cervical in its origin, while the thinner fluid comes from the corporeal endometrium.

Even in cases in which no definite lesion of the tube is left, the prognosis for fertility is always doubtful, yet the percentage of such cases in which fertility is possible is sufficient to warrant the institution of a moderate amount of treatment for sterility in favorable cases, even in the absence of other symptoms, provided the patient is willing to submit to it with the understanding that its issue is doubtful.[9]

The first step in the treatment of such cases consists in the generally somewhat tedious process of disposing of the cervical plug, and the first and most important point in this process lies in ensuring the freest of drainage from the cervix. The cervical canal is frequently somewhat dilated by the thick mucus which it contains, and no permanent recovery can be expected unless the external os is larger than the canal above it. The shape of the canal should be carefully studied out with the sound, and if there is the least constriction of the external opening this should be enlarged on each side laterally with scissors before any other treatment is undertaken (the little snips are painless and may be made without anesthesia). The mucus should then be thoroughly removed from the cervical canal with a dull curette and the surface carefully wiped out with a powerful disinfectant such as iodophenol (tincture of iodine 1 part, crude carbolic acid 2 parts), and this process should be repeated at short intervals until the stringy mucus is no longer formed. These applications are to be directed against the cervical canal alone and should not be allowed to pass through the internal os; but if after the cervical discharge has been rendered approximately normal, that is, no longer semisolid, the uterine discharge is still clouded and overviscous, the patient is in condition for a curettage. This should be as thorough as possible, should be preceded by a dilatation of the cervix so complete as to ensure permanently free drainage, and should not be followed by packing of the uterus. Disinfectant applications to the corporeal endometrium are seldom wise, but the curettage should be preceded and followed by the thorough relief of congestion and the maintenance of the best drainage by continued care of the cervical canal. Success in the management of these difficult cases usually involves also the consideration and care of all the possible complicating causes of sterility, both local and general.

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[9] During the seropurulent condition local treatment of the endometrium of any kind is more likely to result in reinfection and reënlargement of the tubes than to do any good. During the stage in which the discharge is still mucopurulent the advisability of local treatment is very doubtful, and it should be used with great caution. It is only after the thoroughly chronic stage has been reached that local treatment should usually be permitted.

Even when an abnormal uterine discharge is due to lesions of the upper genitalia, the vaginal discharge is soon affected by contact with it; hence the rule that an abnormal uterine discharge of necessity implies a similar vaginal affection, which should be subjected to treatment simultaneously. The converse of this rule, however, is not always true, and those cases in which only the vaginal discharge is affected, though often obstinate, usually yield in the end a very large percentage of success. When the vaginal discharge is of the type to which I have given the provisional name of fermentative, that is, abundant, acid, and yellowish, but not purulent, while the uterine secretion is normal, the frequency of easy relief by antifermentative treatment lends some support to the provisional hypothesis that this form of change is due to an overactivity of the normal flora.

This form of abnormality is always remediable by thorough and repeated disinfection of the vagina; but a great deal of experience in its treatment has convinced me that while this statement is most definitely true, a clinically adequate performance of the disinfection involves an extreme attention to detail, and is seldom accomplished satisfactorily in less than from six to twelve treatments, repeated at intervals of two to three days—it may require more. One detail, which I believe to be especially essential to effective disinfection, is that the vagina during treatment should be so thoroughly distended that the crevices between the rugæ should be thoroughly exposed, and the rugæ themselves as nearly as possible effaced. This involves in most cases a thorough loosening of the garments about the waist and the assumption of the knee-chest position. The disinfectant used should be one which, without excoriating the vagina, is capable of penetrating the superficial epithelium. The text-books on pathology tell us that the vagina is liable to an exfoliative vaginitis in which the superficial epithelium is cast off in a thin eschar which may sometimes be detached in the form of a more or less complete cast of the vagina, and that this condition may be produced by the use of many antiseptic and astringent agents; they do not tell us what the vendors of nostrums have long known, that the separation of these eschars of a mild exfoliative vaginitis is not necessarily attended by any excoriation of the surface, but leaves behind it when produced by most reagents a normal and clean-looking vaginal mucous membrane. A preparation which was at one time exceedingly widely sold was an example of a nostrum which, though not very valuable, achieved its popularity by the mental impression produced by the expulsion after its use of such a cast of the vagina—of course in connection with the highly colored statements as to its nature, which were published with the remedy. The word eschar, however, is overdescriptive of the exfoliation produced by the milder reagents. I was for long uncertain whether it was anything more than a coagulation of the vaginal mucus, but I have become convinced that, which ever it is, it is harmless and an index to an efficient disinfection of the infected vagina.

The technique which, after trying many substances, I have found most effective is, after putting the patient in the knee-chest position and exposing the vagina with a Sims speculum, to dust its walls thoroughly with powdered protargol, through a powder blower, in such quantity that the powder adheres to the wall of the vault over its whole surface; a pledget of dry absorbent cotton is then placed against the powdered surface, the speculum is slightly withdrawn, and the surface which is newly exposed by its withdrawal, as well as those which have been wiped clean by the passage of the cotton, are repowdered, until by a repetition of the process the entire vaginal wall has been covered with the antiseptic. The patient is directed to return at the end of forty-eight hours. She is again placed in the knee-chest position and the cotton is removed, when if the protargol has been thoroughly used, the greater part of the vagina will be covered with a thin, grayish membrane, which can usually be wiped from the surface by the gentle use of absorbent cotton. This eschar is too superficial to be harmful; it will usually be absent from some parts of the vagina, and will perhaps at others be too adherent to be easily removed; no effort is made to detach the latter, but the dressing of protargol and dry cotton is repeated precisely as before, and the process is repeated at intervals of two or three days until the discharge has become scanty and colorless. Aristol is then substituted for the protargol for one or two succeeding treatments, and after an interval of a week or less without treatment the secretion is reinspected and tested for acidity. If it is normal it is reinspected at intervals of a week or more, since the yellow color and overacidity are not infrequently reproduced after an interval without treatment, but they are then usually easily overcome by comparatively few applications. In the absence of an alteration of the cervical discharge, that is, in merely fermentative vaginitis, again to coin a term, this treatment rarely fails to be effective, and permanently effective.

Sterility in women may be due fundamentally to any one of many causes, but almost invariably, if not always, the mechanism of the production of sterility is by a change in the secretions. Its relief is always dependent upon the restoration of the secretions to the normal, but this can rarely if ever be brought about unless by the complete relief of the underlying condition which has caused the change in the secretions.

A vague comprehension of the mechanism by which sterility is produced as a result of the underlying lesion and the merely empirical treatment which is too often applied to it have long caused the subject to be looked upon as one of the opprobia of gynecology. A thorough diagnosis of the whole condition of the patient and more especially of her genital system as a whole at the beginning of the case, and a willingness on the part of the patient and the attendant to pursue care in subsequent treatment into minute detail, will not yield uniform success, but it does raise its percentage to a point at which the treatment of sterility becomes a satisfactory part of practice. At least a fairly accurate prognosis of the percentage of chance for each individual patient can usually be made and submitted to the patient at the beginning of the treatment.

THE INFLUENCE OF TUBERCULOUS ANCESTRY ON THE PROGNOSIS IN PULMONARY TUBERCULOSIS.

By Karl von Ruck, M.D.,
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For centuries the opinion has been generally held that heredity exerts a very important and unfavorable influence on the transmission of, and the prognosis in, pulmonary tuberculosis. So firmly was this idea established that any member of a family, in which tuberculosis had occurred even remotely and who contracted the affection, was often believed to be hopelessly doomed. Thus Morton[1] considered that the disease in hereditarily predisposed subjects was fatal in most cases, the underlying cause not being within the reach of the medical art. In more recent literature this view is still prevalent. Jaccond[2] divides phthisis into three forms, one of which is the hereditary; and this form, from the mere fact of its being hereditary, offers the least prospect for recovery. Yeo[3] makes one of the conditions for cure "the absence of hereditary taint." Ribbert[4] thinks that children with a tuberculous ancestry are not only more disposed to infection, but that the severity of the resulting disease is conditioned by the degree of this predisposition. In further support of this idea Williams[5] shows, from the records of 460 patients, that a definite family history considerably increases the gravity of the prognosis. Of 374 patients who were discharged more or less improved only 29.03 per cent. showed a family history of tuberculosis, while of 86 patients who were not improved, or who had grown worse or had died, 65.03 per cent. had a history of tuberculosis in their families. Riffel[6] asserts that in families in which both parents have a hereditary predisposition, the mortality in most cases is greater than in those in which only one of the parents is so predisposed. In the latter case it makes no difference whether father or mother is affected.

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[1] Phthisiologia, etc. Francofurti et Lipsiæ, 1691, p. 151.

[2] The Curability and Treatment of Phthisis. English by Lubock, New York, 1885, p. 30.

While it thus may happen that tuberculosis appears to be a family trait, and that the disease appears to be "handed down" as it were, from parent to offspring, occasionally perhaps passing over one, or even two, generations, such a history does not *ipso facto* make the existing disease more serious. In fact Reibmayr,[7] although insisting upon the hereditary transmission of the disease, holds that a specific resistance is acquired by the descendants of the tuberculous ancestors which increases in the course of generations and eventually amounts to immunity against the pathological tendency.

For some time various observers have come to the conclusion that the existence of hereditary predisposition being granted, this factor need not necessarily darken the chances for a cure, and that indeed, it might even be a favorable factor in the prognosis. Flick[8] is one of those who deny the influence of heredity from an etiological standpoint and believe in a transmitted partial immunity. Weicker[9] recalls that Meissen was the first one thus to point out that patients with hereditary predisposition are not at a disadvantage in point of the improvement aimed at, as compared with those without hereditary predisposition. This view is also supported by Turban[10] and by Rumpf.[11] The results obtained in enduring improvement by Turban and by Weicker were as follows:

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[3] Brit. Med. Jour., vol. i, 1892, p. 106.

[4] Ueber Vererbung, Marb. Akad. Reden, 1902, No. 6, p. 25.

[5] Diseases of the Chest, London, 1903, p. 10.

[6] Mitteilungen über die Erblichkeit und Infektion der Schwindsucht. Braunschweig, 1892, p. 181.

[7] Die Ehe Tuberkulöser und ihre Folgen, Leipzig and Wien, 1894, pp. 60 ff.

[8] Jour. of Tuberculosis, vol. i, 1899, No. 4, p. 187.

[9] Tuberkulose-Heiltsaetten-Dauererfolge, Leipzig, 1903.

[10] Beitrage zur Kenntniss der Lungentuberkulose, Wiesbaden, 1903, p. 147.

[11] Schroeder and Blumenfeld, Hdb. d. spec. Ther. d. Lungenchwindsucht, Leipzig, 1904, p. 477.

Turban: without hereditary predisposition, 44.8 per cent.; with hereditary predisposition, 49.6 per cent.

Weicker: without hereditary predisposition, 41.1 per cent.; with hereditary predisposition. 46.6 per cent.

Weicker concludes that these patients enjoy a relative degree of immunity, and supports his conclusion by further statistics, from a study of 3299 cases with the following percentages:

			Able to work.	
	Predisposed. Per cent.	Not predisposed. Per cent.	Predisposed. Per cent.	Not predisposed. Per cent.
Cured . . .			64.5	62.0
Improved . .	86.5	81.2	16.4	15.0
Not improved .	11.8	15.3	7.5	9.9
Grown worse .	1.5	2.7	11.4	12.3
Died	0.2	0.8	0.2	0.8

King[12] believes that there is a certain degree of immunity conferred on the children of phthisical parents. He offers the statistics of 242 cases of pulmonary tuberculosis to sustain his opinion, and he shows that tuberculosis occurring in the parents, or a tuberculous family history, not only has no adverse prognostic influence, but that a certain degree of immunity is transmitted from parent to offspring, and he concludes that hereditarily predisposed subjects of phthisis show a sufficiently increased resistance to the progress of the disease to justify him to accept as true a transmission of partial, although not a sufficiently protective, immunity and an increased tendency to recovery.

Turban[13] further shows that when both parents were tuberculous, lasting improvement occurred in 50 per cent. Rumpf[14] has also found that heredity is not a bad factor in the prognosis. He concludes, however, that it will be better for the purpose of prognosis to consider the degree of resistance, rather than the origin of the infection. Cornet[15] considers the clinical results equal for both classes. Solly[13] concludes from his investigations that, with certain exceptions, a family history of phthisis offers no worse, and perhaps a little better, prospect of recovery.

That there may be an increased resistance to tuberculosis in the descendants of tuberculous families, as Reibmayr has suggested, appears to have some confirmation by the results which Petruschky[17] claims from treatment with tuberculin; he states that 54 mild cases, mostly in families with hereditary predisposition, had all practically recovered.

Reibmayr[18] is perhaps the most extreme supporter of the idea of an acquired and transmitted immunity. According to him patients, with one or both parents tuberculous, are not worse off than those whose parents were healthy, for, he says, "the more thorough the tuberculization in the progenitors, the greater is the resistance to the disease in the offspring." He holds, that the disease is transmitted by a specific virus, while at the same time there occurs a transmission of specific resistance. This specific resistance or immunity becomes the more marked and obtains the more rapidly in tuberculous families, and in races and nations, according to the length of time that tuberculosis has been prevalent among them, and according as they intermarry closely, or are isolated geographically; the Jews and people living on islands, as for instance the English, have, for the reasons stated, acquired the greatest resistance. According to the degree of inherited resistance he divides all tuberculous patients into four groups:

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[12] Med. Record, vol. lx, 1901, p. 565, and Trans. Brit. Congr., 1901, London, 1902, vol. iii, p. 611.

[13] Loc. cit.

[14] Loc. cit.

[15] Die Tuberkulose, second ed. Wien, 1907, p. 469.

[16] Trans. Am. Clin. Assoc., vol. xi, 1895, p. 9.

[17] Kriterien und Kontrolle der Heilung bei Lungentuberkulose, 1903.

[18] Loc. cit.

1. *Resistance Low; Prognosis Almost Uniformly Fatal.* The course of the disease in the individuals in this group is more acute, the tendency to recovery is slight. Periods of abatement are short, and therapeutic measures are almost without avail. As a rule, there are many children in the families, premature labors and abortions being frequent. The mortality in children is great, especially during the first years of life. Of ten to twelve children in one family only few grow up, and although these may reach maturity, yet in the end they die of tuberculosis. These latter represent the more resistant members of the family and the duration of their lives depends upon the attention paid to the laws of hygiene.

2. *Resistance Slight.* Childhood mortality is great, but the children live longer than in Group 1. Still they die usually before the tenth year of their age (the few more resistant ones surviving),of scrofula or tuberculosis of the bones and joints. In all cases there is a tendency to abscess formation. Affections of the lungs and pleura are frequent. The survivors, being more resistant, may reach fifty years of age under a hygienic mode of life, but they mostly die between the twentieth and thirtieth year, and as a rule of tuberculosis, or of purulent pleuritic exudates or pneumonia. Those reaching adult life suffer much from bone and joint tuberculosis of the fungoid form. The tendency to abscess formation persists, and indeed predominates. The course of the disease is more chronic than in Group 1. Recovery is still rare; relapses are the rule. Tuberculosis of the lungs still predominates; the tendency to suppuration is well marked.

3. *Resistance Moderate.* The number of children born in this group is less, and deaths under ten years of age are less frequent. While in the preceding groups the scrofulous affections of childhood are torpid, in the present group they are of the erethic type; the children have an anemic appearance; nervous disturbances are more in evidence; there is less tendency to suppuration and a more marked

tendency to recovery from glandular, bone, and joint affections. If suppuration occurs, recovery is rapid. The lungs in members of this group are more resistant, apex catarrhs, and apex infiltrations are still frequent, but the tendency to recover predominates. Individuals reach an age of forty, fifty, or sixty years, and they die less frequently of tuberculosis than those of Group 1 and 2.

4. *Resistance Complete.* The inherited immunity manifests itself to such a degree that, unless it be lowered by an unhygienic mode of life or by other causes, individuals do not acquire tuberculosis. The adults in this group are neurasthenics, but attain a greater age. Their children continue to resist tuberculous infection, unless a union with a non-immune person again reduces the hereditarily transmitted protection.

Reibmayr was not the first to express this idea of an acquired immunity. Rindfleisch[19] declared in 1881 his view, that tuberculosis was inherited like syphilis and that it was at one time a similar infection, only perhaps even more malignant. He thought that by its great and general extension, the whole human race had become permeated with the specific poison which in time had produced a certain degree of immunity.

The statistics published by the German Imperial Health Bureau (Hamel[20]) give data from which the influence of an hereditary predisposition on the prognosis may be estimated, and from their examination I find that 2150 patients (both sexes) were discharged as cured. In the cured cases a history showing the ascendants to have been tuberculous was given, positively in 486 cases (22.60 per cent.), and probably in 588 cases (27.35 per cent.).

Of the remaining 2566 patients (both sexes) who were discharged as not improved, or as having grown worse, the history showed tuberculous ascendants positively in 540 cases (21.05 per cent.), and probably in 611 cases (23.81 per cent.). Taking the "positively" and the "probably" tuberculous ascendants together, we have of the 2150 cured patients 1074 (49.45 per cent.), giving a history of tuberculous ancestry, whereas a like history was shown in 1151 (43.29 per cent.), of 2566 patients, who failed to improve. This shows a difference of 6.16 per cent. in increase of frequency of tuberculous ascendants for the former.

A study of the records of the last 1445 cases treated at the Winyah Sanitarium showed a history of tuberculous ancestry in 487 (31.11 per cent.). In 768 cases discharged apparently cured a history of tuberculous ancestry was recorded in 248 (32.16 per cent.); of 255 cases in the first stage, 80 (31.37 per cent.); of 357 cases in the second stage, 108 (31.20 per cent.); and of 156 cases in the third stage, 60 (39.16 per cent.). In 559 improved cases a history of tuberculous ancestry was recorded in 186 (31.50 per cent.); of 27 cases in the first stage, 8 (29.91 per cent.); of 239 cases in the second stage, 77 (32.62 per cent.); and of 293 cases in the third stage, 101 (34.47 per cent.). In 187 cases that failed to improve, or grew worse, a tuberculous ancestry was recorded in 47 (25.13 per cent.); of 10 cases in the second stage, 2 (20.00 per cent.); and of 177 cases in the third stage, 45 (25.42 per cent.).

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[19] Virchow's Archiv., vol. lxxxv, 1881, p. 71.

[20] Tuberkulose Arbeiten., Berlin, 1904 to 1906.

Taking the 1327 cured and improved cases together, with a tuberculous history in 434 (32.63 per cent.), there appears a difference in frequency between these and the 187 cases in which no improvement resulted, of 7.50 per cent. in favor of hereditary influence from a prognostic standpoint.

It will be seen that the comparative statistics in the cases observed in the Winyah Sanitarium do not differ materially from those supplied by Turban, Weicker, Hamel, and King, and they support the views of Solly, Petruschky, Cornet, and others, who do not believe that a tuberculous ascendancy in itself clouds the prognosis in phthisis. The combined observations deal, however, with too small numbers, and the differences shown are not great enough to accept Reibmayr's extreme position, so elaborately set forth by him in his interesting monograph.

In any case the study of this question is extremely involved and difficult, and requires the consideration of many other influences which affect the course of the disease in the individual patient. In our cases, we have only extended the study to the different stages of the disease, but have not taken into consideration the age of the patient, the particular form of the disease, the presence or absence of tuberculous or other complications, the duration of treatment, and other physical and personal features, which existed and undoubtedly varied greatly in the cases as they are grouped. Such studies, it is hoped, will be undertaken hereafter.

For the present, it is at least comforting to have shown that the indications point in favor of the view that the mere fact of a tuberculous family history being shown in an individual case will not diminish the patient's chances of recovery, and that we may thus not only comfort some of this class of patients and their friends, but also by removing their fears of being at a disadvantage or perhaps doomed to the same fate which had previously overtaken their parents and other relatives, cause them to make more determined and better sustained efforts for their own recovery.

PATHOLOGY OF ADENOIDS AND ADENOID TUBERCULOSIS.[1]

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Although the clinical conditions associated with adenoids and enlarged tonsils are now widely recognized, the pathology of these overgrowths is not fully understood. In the present paper I have attempted to summarize some of the views recently advanced concerning the pathology of these tissues, especially as to their relation to tuberculosis. The subject has been investigated in connection with the study of a series of adenoids primarily with a view to determining the incidence and significance of tuberculosis in these tissues. In these examinations evidence of tuberculosis has been the main object of search, but I have taken the opportunity to note the general histological features which have been the subject of comment by other observers.

The material was obtained from the Nose, Throat, and Ear Department of the Royal Victoria Hospital. Seventy-five adenoids were examined histologically, and in 12 cases this was supplemented by the inoculation of guinea-pigs. In many cases the faucial tonsils were also carefully examined. The study does not represent a consecutive group of cases, as too much material was available and the work was interrupted on several occasions; on the other hand, it is not selective, except in regard to a few of the cases used for inoculation. The examination has been thorough in each case, and I feel that the findings represent a fairly accurate estimate of the incidence of tuberculosis in the cases studied.

Cases of Adenoid Tuberculosis. In 5 cases histological evidence of tuberculosis was found; in 4 of these the disease was clinically primary in the adenoid, in the fifth the adenoid was probably involved secondarily to a pulmonary focus, and in this case the faucial tonsils were also affected. The percentage of primary adenoid tuberculosis found was thus 5.3 per cent. The notes of these cases, together with the one of secondary involvement, are as follows:

Case I.—Male, aged nine years; adenoid removed February, 1905.

Case II.—Female, aged eighteen years; adenoid removed November, 1905. These 2 cases are very similar as to history. Both gave a family history of tuberculosis, but showed no physical signs of tuberculosis either before or after operation (report from family physician, October, 1906). Sections of the adenoids showed typical tubercles with giant cells and caseation (Figs. 1 and 2). Tubercle bacilli were not demonstrated.

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[1] Read at a meeting of the Montreal Medico-Chirurgical Society, February 15, 1907.

Case III.—Male, aged seventeen years; adenoid removed November, 1905. A history of family tuberculosis, and of hip-joint

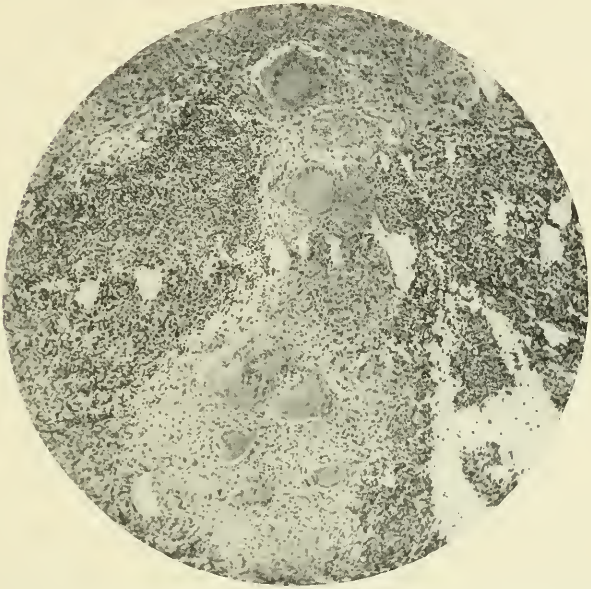

Fig. 1.—Adenoid tuberculosis (Case II). Confluent tubercles with abundant giant cells and caseation. (No. 3 objective.)

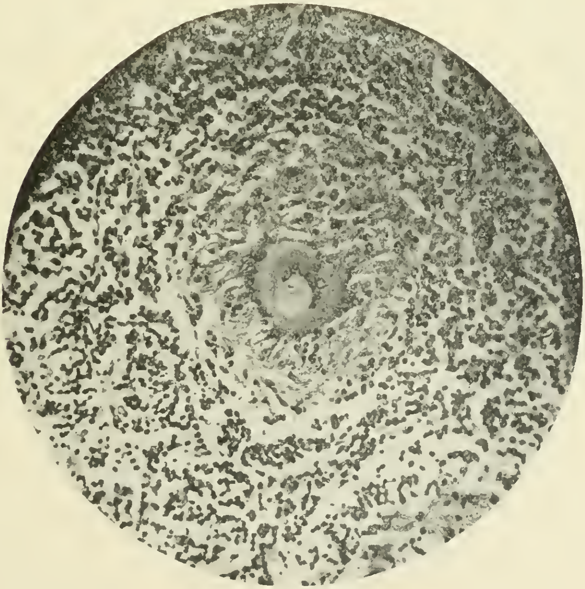

Fig. 2.—Adenoid tuberculosis (Case II). Central giant cell surrounded by a zone of epithelioid cells showing defective staining reaction. At the lower part the edge of a neighboring tubercle is seen. (No 6 objective.)

disease in childhood at age of six years (healed). At present in robust health and free from signs of pulmonary tuberculosis—before and after operation (examined October, 1906). Sections of adenoid showed typical tubercles (Fig. 3).

Case IV.—Female, aged nineteen years; adenoid removed January, 1906. Caseous gland at the angle of the jaw on the left side; otherwise free from signs of tuberculosis, and in robust health. Adenoid showed definite tubercles (Fig. 4), but the tonsils showed no sign of involvement. Subsequent history beyond a few weeks is unavailable, but at that time the patient showed no sign of pulmonary involvement.

In these 4 cases the diagnosis of *primary adenoid tuberculosis* has been made (on clinical evidence).

Case V.—Male, aged twenty-six years; adenoid removed March, 1906. Had a large mass of caseous glands in the left cervical region. Though there were few physical signs of lung involvement, he had a marked tracheitis and slight elevation of temperature. After operation he developed active pulmonary tuberculosis. The adenoid and tonsils both showed tubercles. Diagnosis—secondary adenoid and tonsillar tuberculosis.

Summary of Inoculation Experiments. The number of guinea-pigs inoculated was 12; 5 died of sepsis; of the remaining 7, 2 were inoculated with adenoid showing histological tuberculosis. No guinea-pig developed signs of tuberculosis. It seems likely that either the virulence of the infection was not sufficient to overcome the guinea-pig's resistance, possibly an obsolete tuberculosis, or that since the foci were discrete and scattered a non-tuberculous portion was inoculated.

The Pathology of Adenoids. In discussing the pathology of the adenoid and enlarged tonsils the features which I wish to emphasize are those bearing on the interrelation of the tonsillar tissues and the general health and well-being of the individual: (1) The causes of tonsillar enlargement, and (2) what is known of the function of these tissues which, under certain conditions, may become important channels of infection.

Attention must first be drawn to the fact that the amount of lymphoid tissue normally present in the pharynx is extremely small and analogous to the lymphoid tissue of the small intestine, Peyer's patches. As Bosworth[2] has pointed out, the conventional tonsil of anatomists is really an hypertrophied one and no more normal than an enlarged lymph node elsewhere. On this same ground Jonathan Wright[3] objects to the use of the term "function" in connection with these tissues, for, he maintains, this implies a normal condition of the organ or tissue studied. Of course, most of the tonsils studied by various observers were probably hypertrophied beyond even the degree popularly considered normal; in the present series all were hypertrophied. What modification this may cause in their function is yet to be determined.

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[2] Trans. Amer. Laryng. Assoc., 1901.

[3] New York Med. Jour., 1900.

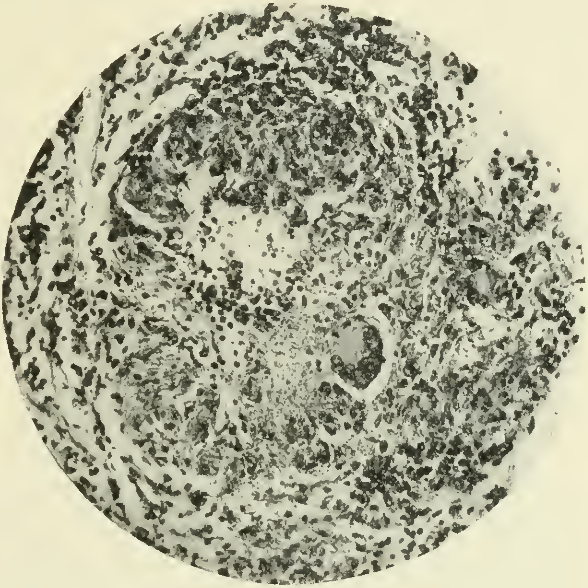

FIG. 3.—Adenoid tuberculosis (Case III). Giant cell and epithelioid proliferation, caseation, and infiltration of the central portion. (No. 6 objective.)

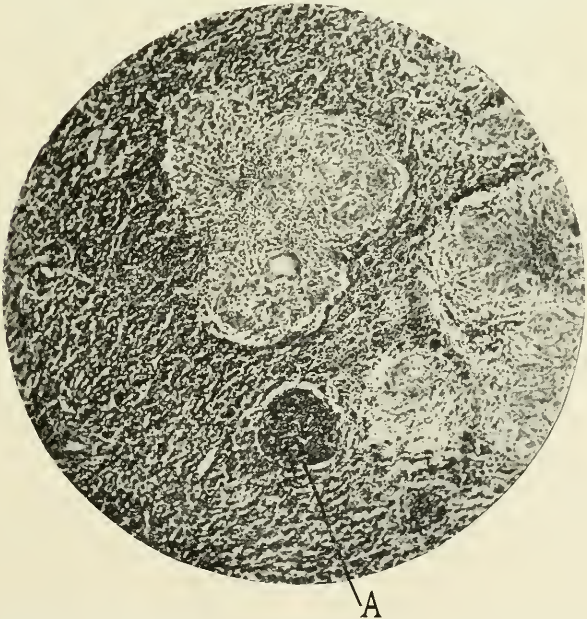

FIG. 4—Adenoid tuberculosis (Case IV). *A*, lymph follicle or germinal centre. The paler areas are tubercles with early caseation. (No. 3 objective.)

ADENOID AND TONSILLAR ENLARGEMENTS. *Histology.* The microscopic appearance of an adenoid or an enlarged tonsil is that of a true hyperplasia, the lymphoid cells being numerically increased. The amount of connective tissue varies to a considerable extent and does not seem to bear any relation to the age of the patient.

Fibrosis was marked in 30 cases of the present series; it was slight or absent in 45 cases. The majority of the cases with fibrosis occurred in the first decade, but marked cases were found in the older subjects. McBride and Turner[4] suggested that the fibrosis frequently seen in adenoids might become a cause of atrophy, but this has not been demonstrated. As Nicholl and Lartigau[5] point out, adenoids which show more or less marked atrophy often lack fibrosis, while others, in which fibrosis is marked, tend to persist. As to the cause of atrophy we know very little, but from the number of adenoids met with in adults, we now know that it is not so constant as was formerly thought.

McBride and Turner[6] point out that atrophy is often inferred from the relief of symptoms as the patient gets older, but that this relief is due rather to the growth of the pharynx making the obstruction relatively less marked than to any true atrophy of the adenoid. It must be emphasized that the most lamentable sequels of adenoids, such as faulty nasal development and chronic nasal or Eustachian catarrh, occur early, during the period of active growth, and that often the adenoid may atrophy only after the harm is done.

The epithelium covering the adenoid is generally columnar, but it is often of transitional or squamous type. In the present series the prevailing type was columnar in 36 cases, squamous in 29, and about evenly mixed in 10 cases. A striking feature of the covering is a rarefaction and lymphoid infiltration of the epithelium. This seems to be constant in the crypts, and very common on the surface as well. This condition has long been commented on and is so extreme at times as to suggest a complete loss of covering. On careful examination, however, the remains of the epithelial cells and the basement membrane can be made out. This change makes the deeper tissue more accessible to invasion by the loss of the mechanical and other protective influences which a healthy mucosa afford. Ewing[7] seems to regard this as an invasion on the part of the lymphoid tissue. Some observers have regarded this condition of the mucosa as evidence of an epithelial origin of lymphocytes. In my opinion it represents merely a chronic inflammatory change in the mucosa, the generative activity of the epithelial cells being directed toward repair rather than to the formation of lymphocytes. A somewhat similar condition is to be seen in the mucosa of polypoidal turbinate bodies. Fig. 5 shows an extreme grade of infiltration of the mucosa and a tuberculous area immediately beneath it, indicating a successful penetration by the tubercle bacilli.

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[4] Edin. Med. Jour., 1897, i.

[5] Amer. Jour. Med. Sci., 1902.

[6] Edin. Med. Jour., 1897, i.

[7] New York Med. Jour., 1897.

THE CAUSES OF ADENOID AND TONSILLAR ENLARGEMENTS. The causes of the lymphoid hyperplasia may be divided into two groups: (1) Constitutional, or predisposing, and (2) local, or exciting. Of these, the predisposing causes are regarded as much the more important.

As regards the frequency of adenoids in Montreal, Birkett[8] in 10,000 cases seen in private practice, found 1605 with adenoids, or 16 per cent. Of course, this percentage is of persons with nose, throat, or ear symptoms. Among persons otherwise healthy Meyer[9] found that of 1000 cases 1 per cent. had adenoids, and Chappel[10] found adenoids in 3 per cent. of 2000 New York school children examined. Sex does not appear to exert any influence.

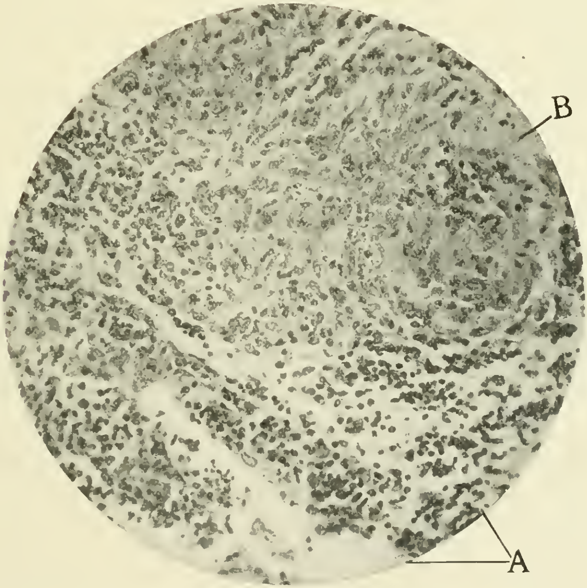

FIG. 5.—Adenoid tuberculosis (Case II). *A*, marked infiltration of the mucosa *B*, tubercle with marked epithelioid proliferation and beginning caseation. (No. 6 objective.)

The age of occurrence in the majority of cases is between the third and fifteenth year. For a short period the greatest number of cases in Birkett's series occurred between the third and fifth year. They are frequently met with up to the twentieth year, and occasionally much later. Cases have been reported in which the condition was found at, or shortly after, birth, but this must be rather rare. Nicholl and Lartigau, in 100 cadavers examined in the New York Foundling Hospital, found no adenoids present under one year.

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[8] Posey and Wright, Diseases of Nose, Throat, and Ear, vol. ii.

[9] Quoted by Nicholl and Lartigau.

[10] Quoted by Nicholl and Lartigau.

This is the more significant when we consider that the type of child in such an institution is one specially liable to these growths, as shown by the great frequency with which they are found between the third and fourth years.

Heredity seems to be an important factor. In Birkett's series there were fifty-three families with two to four children affected. Tuberculosis, syphilis, gout, alcoholism, and old age in the parents, are mentioned as predisposing causes. The acute infectious fevers are also mentioned as antecedents of importance, but Gradenigo[11] states that although adenoids are supposed to follow these conditions, he has not been able to confirm the view by observation. McBride and Turner point out that such cases have seldom been examined for the presence of adenoids. These may have been present before the illness which has merely aggravated the symptoms and thus drawn attention to them.

Hertoghe[12] came to the conclusion that adenoids are a constant symptom in myxœdema, and that those subjects who show adenoids have at least a predisposition to thyroid disturbance. This has not been accepted, and it has been pointed out that their presence has either been a coincidence or is to be explained on the basis of a common etiology, for adenoids occur commonly in districts where thyroid disturbances are very rare.

A view mentioned by Osler[13] and many American writers, is that the enlargement is probably dependent on a disturbance of the general lymphatic system closely allied to, or a mild type of, lymphatism or status lymphaticus. Ewing has published an interesting paper on this condition and its clinical associations, such as rachitis, various anemias, hemophilia, disturbance of the thyroid, and hypoplasia of various organs. He points out that the common factor of importance appears to be a greatly lowered vital resistance from inherited or acquired influences.

This furnishes a common basis of explanation for the very varied etiology and associated conditions—tuberculosis, syphilis, old age, or other conditions in the parents, or an acute illness in the child, being regarded as acting not in any specific way but merely by lowering the general vitality. Friedländer[14] mentions this view as explaining some of the enlarged lymph nodes met with in childhood in other situations. The local causes are regarded merely as contributory and are the various conditions leading to irritation or congestion. Such are, recurrent attacks of acute inflammation, congenital smallness of the pharynx, or some unhealthy condition in the nasal passage or mouth. Local tuberculosis is regarded by most authorities rather as attacking an adenoid already present than as being a cause of enlargement. In many cases no definite cause can be discovered. In certain

[11] Die Hypertrophie der Rachentonsille, Turin, 1901.

[12] Royal Acad. de Med. de Belgique, 26 Mar., 1898. (Quoted by Gradenigo.)

[13] Practice of Medicine.

[14] Arch. Ped., 1902.

cases, contrary to the popular impression, puberty marks a period of increased growth rather than of atrophy.

THE FUNCTIONS OF THE TONSILLAR TISSUES. The earlier writers confined their notice to the faucial tonsils, which was only natural considering the prominent site of these in the pharynx. There seems to have been a popular belief that they had some obscure relation to the genital apparatus, but this does not seem to have ever been seriously advanced. A more academic view was that they secreted mucus to lubricate the food bolus in its passage down the gullet. As soon as histological studies were made, it was pointed out that they were essentially lymphoid in nature, the mucous glands present being connected with the mucous membrane covering it, not part of the tonsil proper. They resemble very closely in structure the ordinary lymphatic gland or node. This demonstration was important in two ways—it classified the tonsils as part of the general lymphatic system, and it led to the grouping of all the lymphoid collections of the pharynx as part of a whole, namely, the tonsillar ring of Waldeyer. This unity has been shown to be not only histological, but also functional. The function of the tonsillar ring may be conveniently considered from two points of view: (1) As regards the activities arising from the nature of its tissues, and (2) as regards those activities associated with location and environment.

The functions of lymphoid tissues are not wholly understood. They are known to be centres of growth for lymphocytes and hyaline mononuclear lymphocytes (Metchnikoff's macrophages). A better recognized function of the lymph glands or nodes is that of filtration of the lymph. They seem to act as sentinels, stopping, or in any case giving warning, of harmful invasion by the afferent lymphatics. Hingston Fox,[15] in 1885, pointed out that far from being secreting organs, the tonsils are really absorbents, and that their spongy structure penetrated by crypts is designed to give a large absorbing surface.

This absorptive function of the tonsillar ring has been established by experimental and clinical observation. Goodale[16] demonstrated the absorption of carmine particles injected into the tonsillar crypts, and Hendleshon[17] independently showed that similar absorption took place when particles were blown upon the adenoid. Wright[18] found a similar absorption of soot particles in adenoids removed from children that had been living in the smoky atmosphere of Pittsburg. Hodenpyl[19] thought that the tonsils had little power of absorption, but his findings do not agree with those of other observers.

The importance of this demonstration arises from the possibilities it opens up of infections of all kinds arising in the pharynx. Ruffer[20] found that absorption of bacteria went on in the tonsils of healthy animals, the presumption being that they were destroyed without injury to the host. This absorption of bacteria without infection of necessity following, has been recognized as occurring elsewhere, especially in the Peyer's patches, and has modified the views generally held with regard to all infections. The matter has been discussed by Adami[21] under the title "Latent or Subinfection."

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[15] Jour. Anat. and Phys., 1885.

[16] Archiv f. Laryng. and Rhin., Band vii., Heft 2 (Quoted).

[17] Ibid., Band viii., Heft 3 (Quoted).

[18] AMER. JOUR. MED. SCI., 1904.

[19] Ibid., 1891.

[20] Brit. Med. Jour., 1890.

In this connection many authorities, notably Gallois,[22] attribute the constitutional symptoms of adenoids to the absorption of various bacteria or their products from the nasopharynx. Boulay and Heckel[23] make the statement that the cases formerly classified as scrofulous or strumous, may now be divided into three groups: (1) The definitely tuberculous; (2) the syphilitic; and (3) the subjects of adenoids, holding that this latter group will account for all cases not included in the other two. They point out that the degree of *adenoidism* in such cases is not at all proportionate to the size of the growth, but depends entirely on the amount of absorption from it. Their views are worthy of attention and furnish a satisfactory explanation of the rapid and marked improvement so often seen in the patient's general condition after an adenoid operation.

The clinical lesson to be learned from all this is that in all cases of cervical adenitis the nasopharynx should undergo careful examination as a probable source of infection. If an adenoid be present it should be removed as a preliminary to other treatment. It is a common experience to have an adenitis of long standing disappear completely after the removal of adenoids or enlarged tonsils. Many of these cases clinically resemble tuberculous adenitis, but are probably only a prolonged irritation of the glands by continued absorption of the various mouth organisms of low virulence.

In acute general infections the tonsillar ring is now recognized as an important channel, and Ullman[24] has collected a large series of cases in which tonsillitis and sore throat have been followed by general infections, such as articular rheumatism, endocarditis, pleurisy, etc., without any other apparent source of infection.

Goodale[25] has discussed the local pathology of these systemic infections. An invasion by a pathological germ is met in the adenoid or tonsil by a local inflammatory reaction similar to that occurring elsewhere. Systemic infection may take place through the lymphatics or blood stream. Infection is favored by the presence of irregular crypts or pockets resulting from previous inflammation or shrinkage of the tonsil. Goodale[26] has recently emphasized this, and points out that question of removal of such a tonsil is to be decided, not at all by its size, which is the popular criterion, but by the presence of irregularities or recesses interfering with good drainage.

[21] Jour. Amer. Med. Assoc., December, 1899.

[22] La scrofule et les infections adenoidiennes, second edition, 1905. (Quoted by Boulay and Heckel.[23])

[23] Archiv. Internat. de Laryng. et Otologie, 1906. Nos. 2 and 3.

[24] Med. News, 1901.

[25] Trans. Amer. Laryng. Assoc., 1902.

[26] Boston Med. and Surg. Jour., September 13, 1906.

TUBERCULOSIS OF THE ADENOID AND TONSILLAR TISSUES. The opinion formerly held that the tonsil and adenoid were rarely affected with tuberculosis, has been shown to be incorrect, though as yet statistics differ as to the frequency and importance of their involvement. This change of opinion has resulted from a more careful and more thorough examination of the tissues. As the type of tuberculosis found is usually proliferative, rather than destructive, the clinical and macroscopic examinations are of little value and the methods of diagnosis are histological examination, inoculation of animals, and examination of sections for the tubercle bacilli.

For an indisputable diagnosis all three methods should have a positive result, but, owing to technical difficulty this is often impossible. It is generally accepted that a typical histological picture is sufficient to establish the diagnosis; that the existence of histologically typical tubercles is to be accepted as proof positive of the presence of the disease; and that the converse proves nothing, that is, absence of tubercles does not surely indicate absence of the tubercle bacilli.

Lewin[27] experimented with tuberculin as a method of diagnosis, but found it unsuitable. He got a severe reaction in many cases, in which the adenoid was not found tuberculous after removal. This is explained by the presence of other latent foci, especially in the peribronchial glands.

An important distinction is to be drawn between the two classes of cases of adenoid or tonsillar tuberculosis, the primary, and the secondary. The primary are such as present no sign of tuberculosis elsewhere which might have been the source of infection, and it is with this class of case that I am chiefly concerned.

The frequency of primary adenoid tuberculosis has varied remarkably in the experience of different observers. McBride and Turner, with histological examination, found 3 cases in 100 examined (3 per cent.), but they considered this too low. Dieulafoy,[28] using inoculation, found 7 cases in 35 examined (20 per cent.); Pluder and Fisher[29] with histological examination, found 5 in 32 cases (15.6 per cent.); Nicholl and Lartigau, by histological and inoculation examination, found 12 in 75 cases (16 per cent.). Wood[30] collected 1671 cases, examined by various methods, and found 88 cases of tuberculosis (5.2 per cent.). This corresponds to the work of Lewin, who found 10 in 200 cases (5 per cent.). In the present series of 75 cases, 4 (5.3 per cent.) were regarded as primarily tuberculous.

Primary adenoid tuberculosis is of importance as a latent focus of tuberculosis not recognizable clinically, which may lead to an infection of the cervical glands, or even to a more generalized tuberclosis. The result of operation in the primary cases is favorable in every way. The site of operation heals as usual. The benefit of removing such a focus is apparent, provided always the focus be primary.

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[27] Frankel's Archiv f. Laryng., 1899. (Quoted by Gradenigo.)

[28] Bull. de l'Acad. de Med., Paris, 1895. (Quoted.)

[29] Archiv. f. Laryng. and Rhin, 1896, Band iv. (Quoted.)

[30] Jour. Amer. Med. Assoc., 1904.

The interest lies not so much in the result of any individual case, as in the demonstration of a primary invasion at this point in the respiratory tract. Although opinion has been strong as to the importance of this channel of infection the amount of positive evidence has been small.

Secondary cases are those which arise in persons with pulmonary tuberculosis, or active tuberculosis elsewhere, and which are regarded as infected by the medium of the sputum or blood stream. This is of interest chiefly as demonstrating the readiness with which tubercle bacilli are taken up by the tonsils when present on the surface. Formerly thought uncommon it has been shown to be very frequent. Walsham[31] examined the tonsils in 34 subjects dead of pulmonary tuberculosis; in only 2 had there been anything to attract attention to the tonsils during life, yet in 20 subjects they were found tuberculous. Wood has collected 136 such cases, including those just mentioned, 94 (69 per cent.) being found tuberculous.

From the frequency of these secondary deposits, it is generally held that before a case may be regarded as primary, the patient must remain free from signs of pulmonary tuberculous for at least a year after removal of the tuberculous adenoid. Cases have been reported of patients, previously regarded as healthy, developing signs of pulmonary tuberculosis shortly after removal of the tonsils and adenoids. The explanation usually given of these cases is that an early or latent focus was already present in the lung at the time of operation, though perhaps escaping notice. The anesthetic and shock of the operation in these cases have probably been sufficient to stir up the activity of the disease. Apart from those in the lungs other foci are not likely to be sources of infection for the adenoid, unless a general dissemination with miliary tuberculosis occurs. The presence of a tuberculous focus elsewhere does not exclude an independent primary infection of the adenoid.

ADENOIDS AND TONSILS AS CHANNELS OF GENERAL INFECTION IN TUBERCULOSIS. The importance of the tonsillar ring as a channel of infection in tuberculosis is determined primarily by the relation which these tissues bear to the tuberculosis so commonly present in the cervical glands. Watson Cheyne[32] considered the infrequency of adenoid or tonsillar tuberculosis as strongly against this channel, and quoted the work of Delepine and Younge[33] to support his view. These observers examined the tonsils of 17 cases of tuberculous cervical adenitis, but did not find any of them tuberculous. He pointed out that, if such absorption goes on, the cervical glands should be very often involved in pulmonary tuberculosis in which the tonsils are bathed in sputum more or less laden with tubercle bacilli. He regarded this as uncommon. Walsham, however, has investigated this latter point with a vastly different result. In 27 cases of pulmonary tuberculosis, he found at autopsy evidence of tuberculosis in the cervical glands, 26 times; the one negative case was one of fibroid phthisis. He points out that the impression of their infrequent involvement must have been based on clinical or macroscopic examination.

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[31] Channels of Infection in Tuberculosis Weber Parkes, Prize Essay, 1903.

[32] Brit Med. Jour., 1899.

[33] Quoted by Moore.

Moore[34] interprets the negative findings of Delepine and Younge quite differently to Cheyne. He regards the absence of tubercles in the tonsils as a sign of failure on their part to react to the infection. He regards this failure to react as evidence of an abnormal patency or "vulnerability of the channels of infection." He regards the finding of tubercles in the adenoid or tonsil as favorable, in so far as it implies an attempt to check the spread of the infection.

The experimental evidence has been clearly in favor of the tonsillar route. Baup[35] induced an experimental tuberculosis of the cervical glands in rabbits by inoculation of the tonsils, usually without a thoracic or abdominal tuberculosis. Wood produced a similar result in hogs. He states that in his opinion, in 90 per cent. of cases of tuberculous cervical adenitis the channel of entrance has been through the adenoid or tonsils. In so far as the cervical glands are concerned, opinion is fairly uniform as to the importance of this channel of entrance. Of course, it is recognized that other channels, such as carious teeth, are to be remembered, and also the possibility of an ascending infection from the peribronchial and mediastinal glands, but these are considered much less frequent.

When we attempt to estimate the importance of this channel in systemic tuberculosis, we find the matter becoming more dfficult and opinions differing widely. It is now recognized that the tubercle bacillus can be taken up without leaving any trace of its passage at the point of entrance. For this reason the matter cannot be reduced to the form of a definite demonstration; there must always remain a certain amount of inference and therefore possible doubt.

The channels of primary systemic infection, which are regarded as of the most importance, are four, given in the ascending order of importance: (1) By the blood—fœtal infection from a diseased placenta; (2) inoculation—by a wound in the skin; (3) by the digestive tract—following the ingestion of tuberculous food, or of tuberculous material introduced into the mouth by the fingers, especially in young children; and (4) by the respiratory tract—following the inhalation of tubercle bacilli with dust. At present we are only concerned with the last mentioned, namely, inhalation.

The present views as to the mode of infection by inhalation are entirely different to those at first advanced in favor of this route. The idea that the bacilli are inhaled into the lung proper is debated, and the peribronchial glands are now regarded as the primary active focus; it is believed that the lungs or pleural cavity are invaded either by direct extension or by spread along the lymphatics of the lung against the lymph current. The frequency of tuberculosis of the peribronchial glands requires mention only to emphasize it. Northrup[36] in 125 autopsies on tuberculous patients at the New York Foundling Hospital, regarded the infection as primary in the peribronchial glands in 88 cases; in 34 cases the primary site was indeterminate, and in 3 cases in the mesenteric glands. Bovaird[37] collected 75 additional cases from the same source, in 60 of which the primary focus was peribronchial and in 15 indeterminate. It has long been recognized that these nodes are also the common site of latent or obsolete tuberculosis in the adult. Loomis[38] examined the peribronchial nodes of 30 adults dying of acute illness or accident while in apparent robust health, and found that in 8 cases they set up tuberculosis in a rabbit. In these cases there was at autopsy no sign of tuberculosis, past or present.

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[34] Jour. Path. and Bact., 1900.

[35] Les Amygdales Porte d'entree de la Tuberculose, Paris. (Geo. Carre and Co., 1900; quoted by Wood.[30])

The point requiring further study is the channel by which the bacilli reach the peribronchial glands. We may conceive of the penetration of the bacilli occurring at any point between the entrance to the nostrils and the bronchi. Experimentally it has been shown that such infection may occur even from the nasal passages. Renshaw[39] introduced tuberculous material into the noses of guinea-pigs without mechanical injury and was able to produce systemic infection through the cervical glands. In no case was the spread to the meninges.

Boulay and Heckel have declared themselves strongly in favor of the view that this penetration takes place in the nasopharynx. They point out that persons of that type of physique, long regarded as especially predisposing to tuberculosis, having the long narrow chest and the characteristic facies, will generally be found to be the subject of adenoids, past or present. They regard the absorption of the tubercle bacilli as occurring, not especially through the adenoid, but from an unhealthy mucosa anywhere in the nasal passages or pharynx; the adenoid, however, presents conditions particularly favorable to such invasion. They call attention to the increasing frequency with which primary tuberculous conditions have been noted in the nose and nasopharynx since the development of rhinology has brought this region within the scope of clinical diagnosis, and in their opinion the lack of autopsy evidence is due to want of routine examination of the nasal chambers at autopsy. They lay stress on the frequency with which pulmonary tuberculosis is preceded by a history of chronic catarrhal inflammation of the nose and throat, with or without an accompanying cervical adenitis. They point out that in certain individuals pulmonary tuberculosis is preceded by a fairly long premonitory period during which, in their opinion, the infection is confined to the glandular system, the rhinocervicomediastinal chain, and that much may be done at this period by treating any possible channel of entrance in the upper air passages, especially an adenoid. They write, of course, on the hypothesis that the nasopharynx is the main channel of infection in pulmonary tuberculosis. It must be remembered, however, that the conditions of the pharynx they describe are usually associated with catarrhal inflammation in the trachea and bronchi, which renders these regions equally open to invasion if infection be presenst; the exposure to infective material, however, is not so great. In nasal respiration the conditions are such that air-borne particles are not likely to penetrate beyond the nasopharynx. It has been shown experimentally that when a current of air is deflected by a moist surface its bacterial contents are deposited on that surface. The deflecting surface in nasal respiration is the nasopharynx. In mouth breathing, the chances of air-borne infection reaching the trachea or bronchi are somewhat greater. An adenoid or nasopharyngeal catarrh may in this way be an indirect cause of invasion in certain cases.

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[36] New York Med. Jour., February 21, 1891.

[37] Ibid., July 1, 1899.

[38] Medical Record, 1890, vol. ii, p. 689.

[39] Jour. Path. and Bact., 1901, p. 142.

A general dissemination with acute miliary tuberculosis may follow the invasion of the cervical glands, but this is a relatively infrequent event.

CONCLUSIONS. 1. Primary tuberculosis occurs in a certain proportion of all cases of adenoids. From the figures of other observers, and my own, this seems to be about 5 per cent. This is regarded as a conservative estimate.

2. In determining the presence of adenoid tuberculosis the histological method is the most satisfactory.

3. Tuberculosis does not appear to be an important factor in the production of adenoid hypertrophy.

4. Adenoids and tonsils are the important channels of infection in tuberculosis of the cervical glands.

5. In the development of pulmonary tuberculosis adenoids may sometimes be direct channels of infection, but their importance is probably more often indirect by predisposing to catarrhal inflammations of the upper respiratory tract.

I wish to express my thanks to Dr. Birkett for the opportunity of doing this work for his department, and for his constant encouragement during its progress; and to Dr. Adami for the opportunity to work in the pathological department and for his advice in preparing this paper. I must also thank Dr. Keenan for his advice and assistance in the pathological work. The photomicrographs were prepared by Mr. Wootton.

CONGENITAL LARYNGEAL STRIDOR.

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Congenital laryngeal stridor has been the subject of many articles, mostly of European source, and of much discussion; the opinions as to its cause are almost as various as the number of observers. It was first described by Rilliet and Barthez, in 1853. In 1877, Krishaber reported 4 cases which existed from birth. The English writers were the earliest to trace its pathology and make the picture complete as it is accepted today. Lees, in 1883, was the first to report an autopsy and show with a plate his finding. In 1884 Gee reported 12 cases of noisy respiration, but the clinical picture given is not always that of a typical case. Politzer in the same year saw a case which he thought arose from vagus irritation, because of noise; he also thought the expiration divided into five or six staccato parts. The absence of dyspnea puzzled him greatly. He gave origin to the nervous theory which has largely obtained to the present time. Then followed cases by Goodhart, Lori, Suckling, Robertson, Smith, Semon, McBride, Variot, Refslund, Sutherland and Lack, Avellis, Herzfeld, Moure, Guinon, Comby, Thomson and Logan Turner, and others. An observation of my own is as follows:

A boy, three weeks old, was seen by me, July 10, 1903, because of a peculiar crowing sound made on inspiration. This noisy breathing was continuous, and accompanied at times by marked cyanosis. It began shortly after birth and went on without any free intervals, causing great distress and much loss of sleep to the parents. To all appearances the child was healthy, with the exception of the efforts made by it to obtain air—which were shown by the inspiratory depression of the jugulum and supraclavicular and infraclavicular fossae, and also by the marked action of the chest. There was no sign of respiratory arrhythmia. The noise was of a crowing, guttural character, and could be heard through two rooms. The cry was normal. He is the fifth child. The family history is excellent. All the other children are healthy.

It was difficult, without an anesthetic, to examine the child with a mirror, owing to the smallness of the parts. Ether was administered, a mouth gag inserted, and a suture put through the tongue which rendered it easy to insert the mirror and see the parts. The epiglottis showed its lateral edges much incurvated, or rather rolled on themselves, so that the lateral edges and the aryepiglottic folds approximated very closely. A small rhomboidal opening could be seen at the base of the epiglottis, which was bounded by part of the lateral borders of the epiglottis and the anterior part of the aryepiglottic folds. It was impossible to see the vocal cords because of the overlying position of the epiglottis on this rhomboidal opening. No external enlargement about the neck or of the jugulum, which would suggest enlarged thymus or thyroid, could be observed.

When about three months old the cyanotic attacks began to disappear, and chest deformity of a marked character became a prominent feature. The costosternal articulations became prominent, and the muscles of the chest somewhat over-developed, giving the appearance of a strong full chest. The marked action of the chest was also noted, and efforts made in inspiration still continued, as shown by the depression in the jugulum and the epigastrium. The sound still continued uninterrupted. When the child was a year old there was apparently little change, except in the tone of the sound, which had become rougher and of a deeper quality. The child looked extremely healthy. Once or twice it had slight colds which lasted for a few days but had no effect on the sound or its character.

When eighteen months old, the child was again examined; the action of the chest was less marked, but still gave the impression of extra effort. The sound was much less marked except on exertion, when its old character would return, but the breathing was always accompanied by a slight stridor. It was now observed that when the child fixed its attention the stridor entirely disappeared, but only for a moment. It was also observed, at this time, that the child's lower jaw did not occlude properly, there being a recession of fully half a centimeter. The child, however, did not breathe through the mouth. When two years of age, the child was examined with a mirror; the epiglottis still retained its peculiar shape, but the interior of the larynx could be seen well. The vocal cords acted normally the tendency of the aryepiglottic folds to approximate was still present. The recession of the jaw was more marked, and the muscles connected with it appeared tense. A slight sound still persisted, but under excitement it would return with some of its old character.

Etiology. Many theories have been advanced as to the cause of this stridor. The first is that of Rilliet and Barthez, that it is due to an inflammation or compression of the trachea, but only one of their cases approaches the real picture of the disease. Lori, of Buda-Pest, followed with a unique theory of clonic spasm of the glottis. He based this opinion on a laryngoscopic view, wherein he claims to have observed the cords pressing against one another. Robertson also favored the nervous origin of the trouble, and thought that adenoids set up a reflex disturbance affecting the abductors. Thomson and Logan Turner favor the nervous theory. They state that the spasm lasts a second or two, and accompanies almost all of the child's inspirations. Sutherland and Lack state that it cannot be definitely decided that the vocal cords play a part, for in some cases there is not an abductor paralysis. Further, it was seen in older patients that the cords moved well and were fully abducted. Yet in these cases stridor occurred on exertion or during a quick inspiration and was invariably associated with a falling inward of the aryepiglottic folds. The nervous theory receives a fatal blow in that the affection persists for months, even during sleep and anesthesia.

Thomson and Turner believe that it is due to ill coördinated spasmodic action of the muscles of respiration, that is, a choreiform respiratory spasm analogous to stammering. They show that the respiration of all young infants is peculiar, in being irregular in rhythm and force, as if the coördination involved in its movements was not under control. These often occur with sudden respiratory efforts, as for example, when a young baby is excited it is likely to make a crowing noise with inspiration, although it does not do it at other times. When coming out of chloroform anesthesia a similar stridor is observed, and a very loud crowing noise is a common accompaniment of operations on the genital organs, due to sudden inspirations which stimulation of nerves of these parts is likely to occasion. Along with the crowing there is also an indrawing of the lower chest wall proportionate to the loudness of the sound. Thomson and Turner conclude that there is a backward development of cortical structures of the brain, which in turn allows incoördination of respiratory movements. They claim that the deformity of the epiglottis is merely an exaggeration of the infantile type and due mainly, if not entirely, to suction of the upper aperture produced by the incoördinated respiratory movements, or, as they call it, stammering respiration. They claim there is no proof that congenital malformation of the larynx exists in these cases. Gay's statement on laryngismus stridulus, to the effect that this is caused by central irritation in children "or better to a deficiency of the higher centres," supports their theory. They hold it is analogous to other coördination neuroses, such as "writer's cramp." Sutherland and Lack oppose this theory of the developmental deficiency of the cortex, their observation showing that the children affected with stridor respire in the same manner as healthy children, that is, with the coördinated and spasmodic type. They also oppose the view that the crowing noise of chloroform anesthesia is due to the same cause as the stridor, because this occurs in adults under similar circumstances.

The adenoid theory was held by many. E. Smith reported 2 cases. The first snored from birth, but this disappeared on operation. The second, which was probably a genuine case, persisted for twelve days after the operation and returned at the end of a year when the child took cold. McBride does not think these cases belong to the genuine stridor group. E. Clarke operated on a patient but the stridor persisted. Variot had a similar experience.

It has been held by some that the sound is produced by tracheal stenosis, caused by enlarged thymus or by enlarged mediastinal glands. The thymus theory was strongly advocated by Avellis, who states that Allen Burns early in the last century thought the thymus a cause of tracheal stenosis and even suggested an operation for it. Siegel had a case which he erroneously diagnosticated as diphtheria. No result followed tracheotomy until the short tube was removed and the long one inserted. The latter caused decubitus. It was then realized that the thymus might be the cause, and Rehn proceeded to carry out Burn's suggestion and opened the mediastinum, lifted the thymus upward, and stitched it to the fascia of the neck. The trouble disappeared. F. Koenig had a like experience by partially removing the thymus.

Avellis speaks of Glockle's observation of 3 cases of stridor which terminated fatally as the result of enlarged thymus. Weigert could demonstrate the compression of the trachea in 1 of these cases. Avellis urges in favor of the thymus theory: (1) The age of the child; (2) cure in the second year; (3) congenital appearance; (4) increased by movement; (5) periodic stopping of the stridor when the position was changed; (6) negative laryngeal examination; (7) high position of the larynx; (8) air current in one bronchus stronger than in the other; and (9) operative results.

Stamm believes the thymus a cause. Hochsinger, of late, has investigated this with the *x*-rays, but his pictures were not looked upon with favor by Ganghofner, under whose suggestion he worked. Siegert also said that often when a large thymus existed he did not observe any stridor and related a case in which enlarged thymus existed without stridor. Thiemich suggests starving the child to produce shrinkage of the thymus, whereupon the child would lose the stridor were this the cause. This treatment has not been followed. Feer, of Basel, observed a case of enlarged thymus with stridor. Hochsinger states that enlarged thymus can exist without stridor.

Tatschner, in an article in answer to Hochsinger, refutes his statements. The patient was his own child, and he showed that by pulling the chin forward the sound disappeared. Whenever he held the child's attention for a minute either by calling it to pronounce words or checking its voluntary activity stridor stopped. It would invariably return, however, when he resumed his play.

As can be seen from the foregoing, the thymus theory is not sufficient, because: (1) The will has no power over the turgescence of an organ controlled wholly by an involuntary innervation. (2) Hochsinger himself admits that in many of his cases, in which he determined excessive hypertrophy, no stridor existed. (3) The *x*-ray picture was proved but once by autopsy. (4) Pulling the chin forward affects the position of the thymus gland, thereby increasing the compression which produces stridor. (5) The stridor observed in compression of the trachea is not inspiratory alone, but is both inspiratory and expiratory.

The case used by Avellis to support his theory, the case of Siegel and Rehn, had sudden attacks of dyspnœa night and day, and was diagnosticated as laryngismus stridulus by M. Schmidt. No specific stridor is mentioned, such as one finds in true congenital stridor. This case was in my opinion a true case of thymus hyperplasia. The child was two and one-half years old when seen, had no history, but began to cough when four or five weeks old. Avellis quotes Schmidt as seeing a patient who "snapped the air," but this has never been observed in true stridor. The attacks of coughing producing cyanosis and suffocating signs are seen in congenital stridor, but the hindrance offered by the malformation of the larynx to the expulsion of mucus sufficiently explains it.

Geffrier brought forth the theory that it was due to enlargement of the tracheobronchial glands. He claimed these could be enlarged at birth and give rise to symptoms which were analogous to those of stridor. This, however, errs in many respects; the noise is both inspiratory and expiratory, cough is similar to that of whooping-cough or measles, and dyspnœa is marked. Then, too, suffocatory crises occur as often as four or five times a day. This enlargement of the glands in children is exceedingly rare and presents a decidedly different aspect from congenital stridor.

PATHOLOGICAL ANATOMY. Lees was the first to show by autopsy the malformation which causes this conditon, and he gives a drawing of the specimen. The patient was a year old when autopsied. It was found that the lateral halves of the epiglottis was so incurvated as to touch each other with their posterior surfaces and almost overlap. The aryepiglottic folds were in close proximity to one another. They were thin and not edematous, the latter fact being important because the child died of diphtheria. There was found between the base of the epiglottis and the approximation of the aryepiglottic folds a rhomboidal opening allowing the passage of air. Lees also mentions a pinhole opening on the superior surface of the epiglottis which allowed some air to enter between the incurved folds of the epiglottis, forming a sort of tube.

Variot, in 1898, autopsied a child twenty-two months old, who died of scarlet fever. He found the epiglottis elongated and the edges approaching each other, and the fibrocartilage thin and supple and of healthy appearance. The aryepiglottic folds were approximated and had to be separated to produce the opening which should exist. This larynx was dissected by Chauveau, who found the arytenoid cartilages longer than usual. The thyroid and cricoid cartilages were normal. It was in this case that he tried intubation and found that the phlange of the tube kept the aryepiglottic folds apart, thus causing the stridor to disappear. Sutherland and Lack confirm with autopsy the above findings. Refslund performed an autopsy on a boy three and one-half months and gives a plate which is similar to those described. The measurements of the parts were:

the epiglottis from base of the tongue to the superior surface 5 mm., from the furrow on the anterior surface to the lateral border 5 mm., or 10 mm. across and 1.5 mm. thick. The furrow was much thinner and transparent than the lateral parts. In this furrow was embedded the connective tissue which forms the median glossoepiglottic ligament coming from the tongue. Owing to the weakness at this point there was great flexibility, hence incurvature was made easier. There was also greater mobility in the epiglottis and the separate halves could be made to overlap easily. Between the arytenoids was a fissure 7 mm. long, formed by the aryepiglottic folds whose edges were close together; from their anterior point of contact to the base of the epiglottis was a separation of 3 mm. Otherwise the larynx was normal.

Thomson and Turner dissected fifty young subjects, some being to the tenth year of life. They found the structures about the vestibule much softer and more collapsible than in the adult, the aperture relatively narrower laterally, owing to the folding backward and inward of the epiglottis and the consequent approximation of the aryepiglottic folds. As the child grows the epiglottis loses its infantile shape and the aryepiglottic folds separate proportionately. They inserted a tube upward in the trachea between the vocal cords and then simulating inspiration, produced the effect of approximating the aryepiglottic folds so as to form a narrow mesial slit. They found that the aryepiglottic folds approximate in infants. They also hold that the peculiar form partially persists up to the seventh year. In this Sutherland and Lack agree.

SYMPTOMATOLOGY. The sound has been variously described by the mothers as being similar to "sound produced by hens," "like a chicken," "like a grunter," "purring like a kitten," and "like croup." Dr. Thomson in his able analysis says, "It begins with a croaking noise and ends with a high-pitched note, and expiration is accompanied by a slight croak when the noise is the loudest. It varies in pitch a great deal. In some cases the note is deep, purring or growling, and in others high and squeaking." The latter holds true mostly in the beginning of the disease, the croaking sound as the child grows older becoming more and more prominent. The loudness varies at times and in almost all of the cases there are short periods of intermission occurring at such times as when the breathing is superficial and regular, or when the child is able to notice by having attention fixed, or when asked to speak. If the child be taken from a cold to a warm room, or when the child is asleep, it diminishes. It increases in loudness either on awakening from a sleep, or on being bathed, or if it is fed too quickly. It can be heard through several rooms of the house; in some instances the observers were attracted by the noisy respiration of the child in the waiting-room of their offices. It appears to be louder when the child is most healthy. When noisiest, it is not continuous, for single normal breaths can be observed. Pauses occur sometimes lasting an hour. They may come without interruption from the noisiest breath and come back also the same way. It often becomes common snoring. It generally has the same timbre and tone-height. Several observers have had the feeling that it was produced by a floating membrane.

There is no difference noted in the sound with the mouth open or shut, or from position. Tatschner observed that on pulling the chin forward it would disappear. In my case pulling the tongue forward under ether did not cause it to disappear.

The stridor increases in loudness for some weeks or months after birth and then continues more or less stationary until the eighth or ninth month. Then it begins to diminish until the eighteenth month. It may last up to the end of the third year. Sometimes it intermits for a month. Even after it has disappeared for a considerable period, if the child takes cold or is out of breath it returns. Occasionally during the third year a short crow or croak is heard. Suckling thought the sound was more intense in sleep and during cry. Lori said in his cases it disappeared for hours between the third and sixth months. He and Refslund observed that it disappeared when the head was depressed or turned to one side; other observers, however, do not bear out this statement.

SIGNS OF OBSTRUCTION. There may be cyanosis at times, but often it is absent. This may be produced as a result of coughing, because of the impediment offered by the deformed epiglottis in expelling secretion. Sutherland and Lack found that in 8 of their cases suffocatory attacks came on when the child was asleep, sometimes when awake. In 1 fatal case observed by them the cyanosis was persistent. When intense the jugulum was tumefied.

The respiratory movements are of a peculiar nature, and at first strongly suggest diphtheria. Most observers have reported increased action of the alæ, marked retraction of the episternal notch, supraclavicular fossæ, and epigastrium, and also some retraction along the ribs, in the axillary region especially. As a result of this, the chest shows gradual prominence of the sternum and the costocartilaginous synostoses, giving in effect a rachitic shape. This has occasioned many errors as to the etiology and diagnosis of the affection. It was thought by more than one observer to be true laryngismus stridulus, this being a frequent concomitant of rickets. This however, can be easily excluded, owing to the absence of the signs, craniotabes, and thickening of the epiphyseal ends. The majority of observers have noted rales all over the lungs, which may be due to the transmission of the laryngeal sound. Thomson states that the lungs are not fully expanded and that breath sounds become feeble toward the bases. This may be disproved because of the intensified action of the respiratory muscles and the absence of signs which would show this condition. Rales and rhonchi are common all over the chest and not in special locations. Postmortem reports do not confirm this, as will be shown under the pathology of the affection.

Variot says that auscultation is of no value, because of noise. Pronounced participation of the abdominal muscles in the respiratory action has been observed. Thomson notes strong action of the sternocleidomastoid muscles. Deformity of the chest persists. Refslund observed movements of the larynx in respiration.

The retraction of the jaw mentioned above in the history of the case is an important factor. It was late in appearance and not observed until the child was over a year old. The retraction caused an occlusion of the teeth of about 0.5 cm. The mouth had a peculiar appearance, yet the child was not a mouth breather. There appears no mention of this symptom in any of the articles published on congenital stridor or on diseases of the thymus, except in one reported by Toilemer, in which stridor ascribed to congenital cardiac malformation existed. He mentions having observed retraction of the jaw of 0.5 cm. His report states that the child was a blue baby and that he worked two hours before the respiration was satisfactory. The presence of this symptom is explained by the action of the muscles attached to the larynx, chin, and hyoid bone, the hyoglossus, geniohyoglossus, digastric, geniohyoid, and omohyoid muscles.

The epiglottis has no special musculature. Luschka states that a small part of the genioglossus muscle is inserted into the ligamentum glosso-epiglotticus medius which has an elevator action of slight development and little importance. These muscles are brought into action when the larynx has a high position, in order to facilitate the separation of the aryepiglottic folds forming the obstruction.

COMPLICATIONS. Most authors insist on the frequency of various pulmonary troubles. Martin observed tracheobronchitis of great tenacity in 1 of his cases. Another child died of bronchopneumonia. Tarny mentions a whooping-cough attack when the child was eleven months old. Guilbert states that bronchitis is frequent, and that one of his patients often had head colds. Rocaz reports a tracheobronchitis in 1 case, in the other 2, bronchopneumonia. One of the latter succumbed. Chauveau's case had bronchopneumonia at eighteen months. In 2 of Thomson's cases dyspepsia was a factor, in a third case the child vomited often and brought up phlegm, in a fourth the child was dyspeptic because the mother did not have sufficient milk, and in the fifth bronchitis was noted. Variot mentions bronchitis in 1, and in another scarlet fever which proved fatal.

Sutherland and Lack had 1 case in which convulsions occurred on the eighth day and recurred up to the second month. One was noted as anemic. Several had bronchitis and 8 had suffocatory attacks. These generally appear within the first two months and are serious. The apnœic condition caused by this peculiar malformation is undoubtedly more frequent than we think. It is not uncommon to hear of newborn children who apparently breathed only with great difficulty at long intervals, sometimes as long as three minutes between each inspiration, accompanied by crowing noise. After a half hour the child would die. Often cases appear in which it is difficult to keep up the respiratory function unless the child is stimulated in various ways, as was observed by Miller, Burral, and others. In these the stridorous breathing continues, but apparently with such effort that the child becomes easily exhausted and dies in a convulsion.

In the first two months of the disease attacks of cyanosis are frequent, but they rarely become so marked as to occasion fear of death. The dyspeptic conditions observed by Thomson and Sutherland, and Lack would seem coincidental.

Suffocatory attacks occur after the first week of the disease, but are of short duration, except in the case of Sutherland and Lack in which convulsions from this source came on every day and lasted two months. Rickets has been mentioned as a common complication. This, however, seems questionable, the error caused by the peculiar conformation of the chest being due to the increased action of the respiratory muscles.

DIFFERENTIAL DIAGNOSIS. At first sight laryngismus stridulus appears to be laryngeal diphtheria, as shown by the retraction symptoms and the noise, but after studying the child a little this can be excluded on the following grounds: (1) There is little or no cyanosis; (2) there are no exaggerated efforts to get air as in diphtheria; (3) as a rule, the history of its early origin is within the first week; and (4) there are no diphtheria bacilli.

Adenoids and enlarged tonsils produce a snoring noise which disappears on closing the alæ. In the cases observed only Clarke and Smith, who held this to be a cause, found adenoids or tonsils.

Diaphragm of the glottis is very rare. Fein could find but 13 good cases and 4 doubtful ones. The symptoms are entirely different in character, the voice being hoarse and dyspnœa observed. Most of the cases occurred later in life, from the seventh to the sixty-fifth year.

Foreign bodies in the larynx must be considered, but here the sudden dyspnœa, convulsive cough, and the phonatory troubles are variable. The history is also valuable in excluding this cause. Semon suggests paralysis of the dilators of the glottis. In this affection the voice is normal, but the dyspnœa intense. The patient's age is also against it.

Tumors of the larynx must be considered. Retropharyngeal abscess is frequent in children but not at birth. This can be excluded by the presence of pain and by digital examination for the swelling. The general condition is bad, as a rule, the temperature is elevated, and the child refuses nourishment.

Hyperplasia of the thymus and enlarged tracheobronchial glands have already been discussed. In such early cases as Miller's, one must consider atelectasis of the lungs, as found in the case he autopsied. He reports one-third of the lung expanded and two-thirds non-crepitant. He also noted that the epiglottis was folded.

Rilliet and Barthez mention atelectasis of one lobe of the lung in a case in which dyspnœa had continued from birth until death at two and one-half months, which was thought at first to be due to stenosis of the trachea.

PROGNOSIS. Sutherland and Lack claim laryngeal stridor to be of little hindrance to growth, except in delicate children. It is bad if pulmonary affections occur. There are in all 101 unquestionable cases reported; death resulted in 21. Lees' patient died of diphtheria, aged thirteen months. Of Refslund's 3 patients, 1 died at six weeks of age, and 1 at nine months, cause not given; the third patient had left lobar pneumonia. Variot's patient died of scarlet fever, aged twenty-two months. Of Sutherland and Lack's 3 patient's, 1 died at the age of two weeks, of convulsions after labored respirations and marked cyanosis, but nothing was found at the autopsy; the second, twenty-five months old, died of "pulmonary disease following a persistent bronchitis" of eight months' duration; the third, an infant of ninteen months, died of tuberculosis. Greene relates the case of a child one year old that died of pneumonia. Both Martin and Rocaz report infants dying of bronchopneumonia at twenty months. Ballin had 3 cases of pulmonary disease, in 2 of which death ensued, 1 a girl two and one-half months old, died of bronchopneumonia (confirmed by autopsy); the second, a boy four months old, had capillary bronchitis (also confirmed by autopsy); the third case, a boy thirteen months old, had pneumonia when nine months old and recovered. Massei reports a boy who died of pneumonia when five months old. Cozzolino observed a boy seven months old with double pneumonia whose stridor disappeared before death.

Miller's 3 cases are especially interesting since death ensued in all within the first twenty-four hours of life. No cry was observed in any of them at birth, and attempts to inspire were irregular and short, accompanied by crowing noise. At times breathing would stop and they became cyanosed. He thought it a spasm, and gave inhalations of ether without effect. Artificial respiration and slapping the back would bring color to the skin. He agrees with Tatschner that the finger behind the jaw would permit respiration to go on easily. The child died apparently of suffocation nineteen hours after birth. On the mother's side, according to her statements, several children died in a like manner a few hours after birth. Burral's patient also died of convulsions two weeks after birth. Levy and Etienne report a boy of seventeen months, who died of œdema of the larynx. Schmiegelow's case is probably one of stridor, with report of a specimen, his pathological diagnosis being "stenosis of the larynx." He gives a good description of the symptoms which are not dissimilar to this affection.

From the foregoing it is to be deduced that death is usually due, directly or indirectly, to the effects of the stenosis. In 7 cases death seemed directly due to the stenosis. After labored breathing for a period varying from a few hours to two weeks the child would become cyanosed and die in convulsions. The next most prevalent cause of death was bronchopneumonia, in 5 cases. There were 3 of lobar pneumonia, and 1 each of tuberculosis, œdema of glottis, diphtheria, and scarlet fever.

Treatment. Tracheotomy and intubation have been tried to alleviate this condition. Variot used the former, but the child failed to retain the tube. However, one important point was gained in that the sound disappeared while the tube was *in situ*. This, as Variot demonstrated postmortem, was produced by the phlange of the tube separating the arytenoids and holding the aryepiglottic folds apart. He advises cutting away the aryepiglottic folds. Ballin did a tracheotomy on one of his patients and the stridor disappeared, but the child died thirty-six hours later of bronchopneumonia.

I think Variot's suggestion to cut away the aryepiglottic folds a good one and should be tried, but in my cases I saw no urgent need of interference and kept the child under observation.

Cutting away the aryepiglottic folds is not a difficult procedure at the present time, owing to recent advances made in endolaryngeal operations by the direct method, especially on children. It is very difficult to operate on a child by the indirect method, because of the smallness of the parts. The introduction of Kirstein's autoscope has rendered the larynx in children very accessible for the cutting instruments. The use of either the Grunwald or the Hajek double curette for working on the sides of the larynx would render the removal of the aryepiglottic folds a very simple matter. The removal of the epiglottis would require a stronger instrument, such as a small-sized tonsil punch. We know that the removal of the epiglottis is compensated for, and no danger can arise because of its removal. The cutting away of the epiglottic folds might be tried first. If this did not give sufficient relief, a fact which could be observed during the operation, the removal of the incurved part of the epiglottis probably would. I do not hesitate to advise this, because there is a minimum of risk.

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WHEN SHALL WE ADVISE TYMPANOMASTOID EXENTERATION IN THE TREATMENT OF SUPPURATIVE OTITIS MEDIA, AND IN WHAT PERCENTAGE OF CASES MAY WE EXPECT A CURE?[1]

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During recent years the prognosis of suppurative middle-ear disease, including all of its complications and eventuations, has very materially improved. A better realization of the importance of the disease has led to its earlier and more persistent treatment, and antiseptic surgery has enabled the aurist to save the lives of many persons whose conditions a few years since would have been looked upon as hopeless. Of perhaps more importance is the fact that surgical improvements have put it in the power of the aurist to prevent many of the serious complications of this disease.

A purulent inflammation of the middle ear may persist for many years without causing serious disturbance of health or destruction of any tissue beyond the confines of the tympanic cavity. On the other hand, it is becoming generally recognized now that the mere existance of such a purulent focus is a constant menace to the health and life of the individual bearing it, and that at any moment it may flare up into an active process which will be extremely difficult to check or control.

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I suppose it may be taken for granted that this Association would recommend the persistent treatment of every case of purulent otitis media until cured, employing the simplest measures possible as long as they give promise of effective results, but resorting to surgical intervention whenever required to overcome an obstinate otorrhœa. Without at all exaggerating the possibility of dangerous complications or sequels ensuing from direct extension of suppuration, from the tympanum to neighboring vital structures, we are safe in saying that no person who is the subject of a purulent discharge from the middle ear should be permitted to look upon his condition as one without danger to life. Aside from the risk of the graver consequences attending purulent invasion of the mastoid process, the soft tissues of the neck, or the cranial cavity, we must remember that the victim of chronic otorrhœa is suffering a steady drain upon his supply of vitality and that in addition to this fixed charge against his resources he carries an undeterminable, yet constant, risk of secondary personal infection and an incubator for bacteria to supply contamination to his neighbors.

It becomes our duty, therefore, to teach the family physician and the public to recognize the fact that purulent otorrhœa is a symptom of a disease which has a most important bearing upon the patient's continued enjoyment of health and life; that it demands immediate and continuous treatment until a cure results, inasmuch as it is susceptible of cure in almost every instance; and that, while we sometimes speak of the measure which is employed as a last resort in the treatment as "a radical operation," it is not *radicalism* but *conservatism*, which demands surgical intervention in chronic suppurative otitis media when other means of treatment have failed of effect.

The prognosis of suppurative otitis media, when properly treated, is extremely good. The majority of all acute cases respond promptly to proper local medicinal measures. By such treatment is meant simply the procuring of free drainage of excretions from the tympanum and the maintenance of as perfect cleanliness of that cavity as is possible. But, in spite of the early institution of skilful treatment, some cases appear to be in the very nature of things destined to pursue a chronic course. This tendency to chronicity may be the result of excessive activity on the part of the infective agent, of inability on the part of the patient to respond to reparative processes, or of peculiar anatomical conditions or pathological alterations which favor the extension or persistence of the necrosis. General hygienic care and tonic treatment, supported by patient, well-directed, and persevering efforts to sterilize the tympanum, will render the invading foe innocuous and bring about a cure in the large majority of this class of cases.

Neglect is perhaps the greatest factor in the causation of chronic purulent otitis media. Ignorance of the character and dangers of the disease, reliance upon home treatment, carelessness in the employment of remedies and measures properly recommended by the physician, or the use of agents better fitted to excite than to allay purulent inflammation, are among the factors we frequently have to blame for the existence of a chronic otorrhœa when first it comes under our notice. But even these chronic cases have a very favorable prognosis when brought under proper treatment, for it has been demonstrated that taking them as they come, and including cases of many years' standing, fully one-half will recover under simple drainage and antisepsis.

Something less than 50 per cent., then, of the chronic suppurative otitis medias will require the consideration of some more energetic measures, such as surgical assistance, for the removal of polypoid tissue or necrotic ossicles through the external auditory canal, or the more extensive surgical intervention embodied in the so-called radical operation. Ossiculectomy, intratympanic curettage, occlusion of the tympanopharyngeal tube by a flap from the tympanic membrane, and removal of the tympanic scute through the external canal are simpler surgical measures which promise satisfactory results in some cases. It is surprising what a large percentage of the very worst-looking cases will respond satisfactorily to the exhaustive employment of painstaking measures to cleanse the tympanum of debris and free it of infective material. If due care is exercised in diagnosticating the factors accountable for chronicity in each case and conscientious efforts made to remove or overcome them, efforts which shall be continued patiently and perseveringly over a period of several weeks or months if necessary, according to the circumstances attending the particular case, I believe but a small minority of even the chronic otorrhœas will require the operation of tympanomastoid exenteration. It seems to me this operation is properly to be held in reserve as a last resort for those cases which do not succumb to milder forms of treatment, but that, while an honest effort to procure healing by simple measures should precede consideration of surgical attention, indefinite prolongation of such treatment is not justifiable; and the fact that serious complications have not supervened does not excuse interminable delay. Not all cases are cured even by the radical operation, but the vast majority are; and so, only an insignificant percentage of the whole number of otorrhœas is left unhealed after thorough treatment. That this small proportion will steadily decrease in the future as the technique of the major operation improves in the hands of the mass of operators, is to be expected.

Nothing has been said regarding those cases of either acute or chronic otitis media in which positive evidences of mastoid or cranial invasion are present, it being assumed that all are agreed upon the necessity for prompt operative intervention as the only safe form of treatment, and that the operation of complete tympanomastoid exenteration should be performed if necessary in order to ablate the disease. It is the object of this paper to consider more particularly that large group of chronic suppurative otitis medias in which the predominant symptom is otorrhœa, and in which no serious complications have arisen. Concerning these cases, if we admit the fairness of the prognosis stated above and accept, as a foundation for determining the treatment to be selected, the belief that while most cases can be cured by drainage, antisepsis, and middle-ear operations a small number will require the major operation, and that this operation is not only a justifiable procedure, but should, as a rule, be employed rather than permit the disease to continue, it becomes necessary to consider the following questions: (1) What are the indications that justify tympanomastoid exenteration? (2) What are the dangers and risks of the operation as compared with the disease untreated? (3) What may we safely say to the patient regarding relief from the otorrhœa and the restoration of function?

1. *What are the indications for tympanomastoid exenteration in the treatment of chronic suppurative otitis media, without evidences of mastoid or cerebral complications?*

Purulent otorrhœa is, perhaps, the single symptom complained of, and, assuming now that other forms of treatment have been properly tried and have failed to control this discharge, we must be guided by a study of the excretion, the determination of the pathological lesion within the tympanum, and the patient's general physical condition. Ascertaining the nature of the bacteria present in the pus affords very little if any assistance, but such investigation should always be made. As a rule the infection is a mixed one. When streptococci are present in considerable numbers we may bear in mind the especially virulent character of this organism, but it is not safe in any given case to look upon the diplococci or staphylococci as less dangerous. I have seen a fatal result in one case of pure infection by Staphylococcus albus, an organism that is looked upon as comparatively innocent. Of much more importance is the discovery of bone-dust or epithelial cells in the excretion. If the washings from the tympanic cavity be centrifugalized and the sediment, under microscopic examination, shows particles of bone structure, we have positive evidence of bone destruction going on, and this is one of the strongest reasons that can be urged for operation. Likewise, the finding of epithelial cells in the pus or in the washings warns us of the possible presence or formation of cholesteatomatous masses, and, if in addition cholesterin crystals are discovered, we have positive assurance of the existence of a dangerous element. These features of the microscopic investigation are the most important single elements in determining the advisability of surgical intervention.

As regards the gross pathological changes in the tympanum, very little information of prognostic value can be gained from their inspection. Lesions of the internal wall can rarely be made out because the disease, when it does spread in that direction, most commonly passes through the round window or, less frequently, through the oval window after erosion of the foot-plate of the stapes. Pus exuding from the oval window or from the promontory, particularly if it rapidly reappears after being wiped away, is positively diagnostic of labyrinthine invasion, and the appearance of granulation tissue springing from the inner tympanic wall, with roughened carious bone underneath, is characteristic of necrosis of the labyrinthine capsule. Tinnitus and vertigo accompanying purulent otitis media, with or without the objective evidences of labyrinthine disease just referred to, while not affording conclusive proof of the extension of purulency, are sufficiently suggestive to justify an exploratory operation and this should always be in the nature of a complete tympanomastoid exenteration. When dealing with large polyps which protrude into the external auditory canal we may well consider the advisability of attempting their removal by a simple snaring operation, to be followed by appropriate local treatment, or the performance at once of the radical operation. Such growths almost invariably spring from an area of carious bone the extent of which disease cannot be prejudged. Forcible evulsion has not infrequently been followed by serious complications; the tearing away of a sequestrum, opening up healthy tissue to the pyogenic organisms which seem to become remarkably active after perhaps a long period of lethargy. If it appears that the polyp has its attachment to the roof of the tympanum or its internal wall, it is probably wiser either to make its removal part of an exenteration operation or to inform the patient that such an operation may soon become necessary. I am, personally, inclined to look upon a middle-ear polyp, whose base I cannot accurately decide to be situated in a comparatively innocent site, as sufficient reason for recommending the radical operation. When a polyp is removed in this way the operation can be completed satisfactorily, the entire suppurative process probably be disposed of at the same time, and the operator has better control of the situation for the prevention of future complications.

Another visible evidence of extratympanic purulency is the finding of pus trickling down over the internal wall from the region of the additus ad antrum. This affords a strong inference of antral infection, and as irrigation is presumed herein to have failed, proper drainage can be secured only by operation through the mastoid. In those cases of suppurative otitis media in which the ossicles are still present, though possibly diseased, we have naturally to consider the relative merits of middle-ear operations through the canal and of complete exenteration. It is scarcely within the province of this paper to discuss that matter, though some mention of it must be made. It was understood as a premise that, whenever clearly applicable, simple middle-ear operations would be given preference over the more extensive major procedure. Naturally there will arise a series of cases wherein opinions would differ as to the applicability of the several operations. I can imagine one surgeon advocating radical measures while another of equal ability and professional prominence would in the same case prefer to try, first at least, ossiculectomy. It is conceivable also that the latter might secure a cure in some of these cases. Consequently there is room for much honest difference of opinion in the selection of cases. The conservative ground to occupy would seem to be a preference for the radical operation only when granulomas, epithelial cells or bone-dust in the pus, or clinical symptoms of extension of the disease beyond the confines of the tympanic cavity point to the necessity for some operation more thorough than can be performed through the canal.

Occasionally the patient's general state of health may be a determining factor in the decision to operate. Just how much influence a chronic purulent otitis may be exerting upon the general system it is always difficult to say, but it is reasonable to suppose that sufficient toxins may be absorbed from the tympanic disease to prove a seriously disturbing obstacle to the growth and development of a child or to cause a decline in the vitality of an adult. It has seemed to me that this possibility has too seldom received its meed of consideration. When considering the indications for operation I should be, therefore, more strongly inclined toward intervention if the patient's general physical condition was below par than if the patient were robust, other things being equal.

2. *What is the prognosis of the operation itself? What are the possible dangers of the operation? How do these risks compare with those of the neglected disease?*

Under the head of prognosis we should consider: (*a*) The question of mortality; (*b*) deformity of the face; (*c*) failure to cure the condition for which the operation is performed, and (*d*) further impairment of the hearing.

(*a*) Any operation which requires the preliminary institution of anesthesia is, of course, attended by the possible mortality of the particular anesthetic employed. As anesthetics are given today in the best hospitals, this danger is reduced to the lowest calculable point, and is so small as be practically a negligible quantity; though the patient or his guardians should always be informed that death from anesthesia is a possibility. In view of the fact that it is generally impossible prior to operation to ascertain with any degree of accuracy the extent of the necrosis, and recognizing also the fact that some of the most careful clinical observers have disclosed at operation complicating lesions which were entirely unsuspected, we must admit the possibility of some few fatal results from the excitation of latent purulent foci or from the coincidence of having operated at the moment when an otitis media was breaking into vital structures. But has the operation any mortality *per se?* Theoretically, one would be inclined to answer this question in the negative, but, practically, it seems impossible to do so. The region attacked is one where a slight error in technique may be followed by serious consequences, and few of us will claim infallibility. A very small number of cases, yet sufficient to demand consideration, have been reported in which death followed the operation without explainable cause. At the 1904 meeting of the American Otological Society, Jack reported a death after operation, for the relief of otorrhœa, upon a particularly robust and healthy man, and Harris spoke of a similar instance occurring in the practice of Dr. Toeplitz. The fact that but few fatal results have found their way into otological literature does not prove their infrequency, but there is no apparent reason for assuming, as some have, that a sufficient number of fatalities have occurred to give the operation an appreciable mortality.

Zeroni,[2] in a most interesting and instructive article on Postoperative Meningitis, reports a critical analysis of 40 cases of death following this operation for the cure of otorrhœa, which he has been able to find in German literature. Careful postmortem examination showed that in 29 there had existed an unsuspected labyrinthine disease, in 10 there was a latent meningitis or extradural abscess, and 1 remained unexplained. This argues the necessity for a more careful search for these deep-seated conditions in cases which we propose to submit to operation. He believes the operation less dangerous than the disease, because a study of these autopsy materials convinced him that most of the cases were steadily progressing to a serious termination and would have inevitably ended fatally had no operation been performed.

(*b*) Since almost all operators now seek primary closure of the mastoid wound in this operation we may pass any consideration of the deformity from postaural openings. The only deformity of an annoying character that may result is that of facial paralysis. Even the most skilful operator, taking every precaution to protect the nerve will occasionally have a paralysis following this operation. When such an unfortunate sequel results we should be slow to impute carelessness or clumsiness to the surgeon. The mere exposure of the nerve sheath to the atmosphere, to infection, or to pressure from the dressings may cause a neuritis or a paresis, and I believe that is often the explanation of those cases in which the appearance of the paralysis is delayed for from twenty-four to forty-eight hours after the operation. Touching the nerve with the instruments is, of course, the most probable cause of the majority of these accidents, but unless the nerve is grossly injured the chances of recovery are good. Complete severance of the nerve or gouging out a portion of it may result in permanent loss of function. The simpler traumatisms or irritations produce only temporary loss of power. In spite of the belief in a good prognosis, one nevertheless views a postoperative facial paralysis with distress and apprehension.

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[2] Archiv f. Ohrenheilk., December 15, 1905, p. 199.

(*c*) Complete cure of the suppurative otitis media does not always result from a satisfactory exenteration. With the best and most experienced operators there still remains a high percentage of failures to cure otorrhœa by this operation. Satisfactory statistics are difficult to arrive at, partly on account of the varying basis on which the indications for the operation are placed. Thus the surgeon who employs the operation for nearly every case of chronic suppurative otitis may be expected to show a higher percentage of cures than one who performs exenteration only in those cases that have been first subjected to every other form of treatment without avail. It may be said also that so far there have been very few statistics published from which conclusions might be drawn. Some of the most prominent surgeons making such reports, and who probably operate only when other forms of treatment would not promise good results, are the following: Stacke has reported 37 cases operated upon with 27 cures (73 per cent.); Panse 57 cases with 31 cures (54 per cent.); Grunert 43 cases and 24 cures (55 per cent.); Poli 40 cases with 32 cures (80 per cent.); and I recently heard Dr. Dench say, in a discussion of this subject, that while in the first hundred cases he had only about 70 per cent. of successes, in a second series of cases about 80 per cent. of cures had resulted.

(*d*) The effect upon the hearing is a question invariably asked by the patient, but I am not sure as to how much consideration it deserves. Conservation of the hearing or of a fractional portion of it has a different value for different individuals. A great many people might be deprived entirely of the function of one ear without being seriously handicapped in their business or social relations. On the other hand, persons whose condition demands acute hearing power can ill afford to lose any or, having lost some by disease, must avoid risk of its further diminution; particularly is this true when both ears are involved. We have all seen cases of extensive destruction of the membrane and ossicles, with persistent otorrhœa, in which the hearing remained nearly normal. In such a case, unless there is some urgent reason for operating, it is quite proper to estimate the possibilities of further impairing the function of the organ. In the attitude I have taken, of recommending operation only in those cases of extensive middle-ear disease, in which other treatment has failed, the hearing power is usually a negligible quantity and occupies a minor place in the consideration of operative treatment.

A few operators have claimed remarkable improvement of hearing following operation in some cases, but as nearly as I can estimate it we are not, as a rule, justified in promising or expecting improvement in the hearing and in many cases will find that it is impaired by the operation. Should we be so fortunate as to eradicate the disease and secure a healthy skin lining for the new cavity formed by the junction of the antrum, tympanum, and external auditory canal it is quite probable that in the majority of instances this very process of epidermization, which contributes to the success of the operation, adds something to the impairment of motion of the stapes. The better the mobility of that ossicle prior to operation the more likely is it to suffer from this cause. It is perhaps only in those cases in which the stapes is firmly fixed by fibrous tissue that the operation improves its ability to respond to sound waves, by liberation from the restricting bands.

How do the risks of the operation compare with those of the disease? It is impossible to make a satisfactory comparison inasmuch as accurate statistics are not obtainable on either side. I have stated my belief that the operation is not very dangerous to life and that most of the deaths that have followed the operation have occurred from some complication previously existing, though unsuspected, rather than as a direct result thereof. Neither can the dangers of the disease be stated explicitly in figures. We all know, however, that the majority of purulent affections of the brain have their origin in neglected otitis media and that many cases of infective enteritis and fatal bronchopneumonias, especially in children, originate from the same source. A most significant fact to be remembered in this connection is that no reputable life-insurance company will take a risk upon an individual who is or has recently been the subject of a purulent otorrhœa. The business man's estimate of such risks is worthy of consideration, at least it may have more weight with the lay mind than any medical argument. In a very instructive paper by Whitehead[3] on the complications of temporal bone disease, based upon a study of the work performed at the General Infirmary at Leeds, England, during the last fifteen years, I find the following significant statement: "There was an antecedent history of chronic otorrhœa in 21 out of the 33 cases of meningitis; in 17 out of the 20 cases of sinus thrombosis; in 20 out of 21 cases of cerebral abscess; in 13 out of the 15 cases of cerebellar abscess, and in 1 out of the 2 cases of combined cerebral and cerebellar abscess." This comes from a general hospital where patients were not admitted previously because of ear disease.

In conclusion, viewing the question from these various standpoints, I would formulate the following rules for guidance when considering the treatment of persistent chronic suppurative otitis media:

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[3] Archives of Otology, 1906, xxxv, No. 5.

1. Broadly speaking, practically every case of suppurative otitis media is assumed to be susceptible of cure by one means or another.

2. Every case of chronic suppurative otitis media, without symptoms of intracranial invasion, should be treated patiently and persistently for a reasonable length of time, but not indefinitely, by well-directed efforts at cleanliness and antisepsis through the external auditory canal. When it becomes evident that these simple measures or minor operations cannot cure the disease, tympanomastoid exenteration should be advised unless in a given case there exists some special reason of socio-economical character that justifies delay and the risks of the disease.

3. The clinical evidences of an inveterate purulency that may help one to decide the question of when to recommend operation are: the finding of cholesteatomatous masses, epithelial cells, or bone-dust in the washings from the middle ear, the tracing of the source of pus to the mastoid antrum or labyrinthine capsule, or the existence of granulomas springing from carious areas of the tympanic wall which cannot be directly inspected and treated.

4. The possible dangers of the operation are believed to be far less than those of the disease.

5. The patient should be told that not every case is curable, even by an operation (the percentage of cures in the obstinately chronic cases probably approximating 70 per cent.), that the hearing power will probably not be improved and may be somewhat impaired, but, that the serious nature of his disease warrants surgical intervention as a prophylactic measure.

6. Every patient upon whom an operation of tympanomastoid exenteration is contemplated should be most carefully studied for a considerable period of time, in order that the slightest evidence of latent meningitis or purulent labyrinthitis may be detected, and when there exists any reason to suppose that the disease has extended beyond the bounds of the tympanic cavity the patient or his guardians should be told that an element of danger attends the operation. The possibility of postoperative complications cannot be ignored and the surgeon must safeguard himself.

SOME CAUSES OF FAILURE IN PLASTIC OPERATIONS ON THE FEMALE GENITALIA.[1]

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Every gynecologist who has opportunities in public and private practise to review his own work and to see that of others, realizes that a very fair proportion of patients upon whom a plastic operation has been performed present either functional or anatomical failure. It is much fairer to our patients, and more just to ourselves, to be self-critical in this matter, than to ignore the fact or to be unduly hypercritical of the work of our colleagues. It is to a consideration of the causes of this failure that I invite attention.

What constitutes a failure in the result of a plastic operation? It may be an *anatomical* failure, a *functional* failure, or a *symptomatic* failure. Perfectly adjusted and united wounds may fail to relieve a woman of her backache or bearing-down pain, because a curettage was omitted or because a heavy, metritic uterus still lies retroverted or retroflexed; or, union may be incomplete or entirely lacking, with the result of a continuance of the symptoms of which the patient originally complained. Again, the restoration of a cystocele or a perineum may be perfect as far as anatomical and functional results are concerned, while the nervous, excitable, irritable woman may continue indefinitely to complain of her pain, her backache, her bearing-down pain, and the long train of symptoms accompanying a nerve wreck. It is, therefore, plain that three important elements enter into the performance of a plastic operation: the patient, the operator, and the technique of the operation, including the after-treatment.

The Patient. We are all too prone to decide, when a woman comes to us with a cystocele, a rectocele, a laceration of the perineum, with symptoms corresponding to those we have learned to attach to these conditions, that operative measures will give her relief. I do not contend that this is not so in the great majority of cases; but we have a further duty than merely to substantiate our diagnosis. We must enter into the daily life of this woman, scrutinizing as carefully as we may her domestic and marital affairs, her relations with those with whom she comes into daily contact, her physical and mental life, and their abnormalities. In other words, we must learn to know the woman as well as we try to know the case. I know this is trite, but it is surprising how many times these elemental yet important factors are overlooked; and in no field of surgery is their recognition more vital than in that which has to do with unfortunate, incomplete, or ignorant obstetrics.

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[1] Read at a meeting of the Obstetrical Section of the New York Academy of Medicine, February 28, 1907.

The selection of patients for plastic operations, then, is the first important step. Those suffering from serious constitutional disease should be treated, as far as possible and consistent with comfort to them, by means other than surgical. Diabetes, chronic nephritis, exophthalmic goitre, active tuberculosis, are contra-indications to operation, except in cases of rectal or vesical fistula, or complete lacerations of the perineum, which threaten the patient's life by sepsis or make her a mere living burden. Even these operations, and the other plastic procedures, are likely to result in total failure or even in death if undertaken in the face of these systemic diseases. We are occasionally compelled, however, to intervene, because of the patient's constant or increasing misery.

Aged women, who can be made comfortable with the aid of a pessary are also better treated thus, than if they are subjected to operative intervention. I have been treating a woman, now seventy-eight years of age, for five years by this means, with perfect satisfaction, the complete prolapse from which she suffers being perfectly controlled. The reason for careful consideration in the case of the aged lies in the greater possibility of postoperative accidents which may befall them: pulmonary embolism, thrombosis, or apoplectic seizures. It is small comfort to have a large cystocele or rectocele perfectly healed, if we have the patient die three weeks after the operation of pulmonary embolism, as happened in one of my cases.

Still another contra-indication on the part of the patient lies in the short time which may have elapsed since labor. Unless a perineal laceration can be repaired within a few hours or a few days after the birth of the child, it is far better, in my judgment, to wait a full eight months. Before this time the tissues are not only still exceedingly vascular, making hemostasis difficult, but they are very brittle as well. Denudation is thus made difficult, and the mucosa tears off in strips, instead of being easily peeled off. I had this experience a year ago, desisted from the operation, and did it four months later without difficulty. Sippel[2] has also recently reported two similar instances, and although these are unique accidents in his experience, he urges that we should no longer accept six weeks' postpartum as the selected time for the performance of a secondary plastic operation, but should await a complete involution of the mucous membrane.

Finally, women who are in what we call a "run-down" condition, whether from too frequent births, too constant care of children, domestic infelicity, household cares, or what not, should first be subjected to the proper sort of tonic treatment. Their bowels, their sleep, their appetite, their exercise should be judiciously prescribed for. Brown has demonstrated favorable results from operations on the insane, and apparently these unfortunates offer no obstacle to a good result. But the familiar type of woman whom I have described will not be a good advocate of plastic operations; for no matter what operative skill may be employed, or what perfect anatomical result may be achieved, the functional result is not likely to be gratifying either to the patient or the surgeon.

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[2] Zentralbl. f. Gynak., 1906, No. 36, p. 1002.

The Operator. In a general way it may be said that plastic operations upon the vagina should be undertaken only by those thoroughly familiar with gynecological work. It is only the gynecologist who knows thoroughly the relations existing between diseases of the uterus and its appendages and the various lacerations of the vagina and perineum. The general surgeon may be perfectly capable of going through the technique of a plastic operation, but unless his training has been as well along gynecological lines—which is rare—he will be more likely to operate when the gynecologist will withhold his ardor, and vice versa. To overlook a unilateral or bilateral disease of the appendages while repairing a torn cervix or performing an anterior colporrhaphy is a technical error which a perfectly performed operation cannot correct. Such a case has very lately come under my observation. On the other hand, the gynecologist can prepare himself for his plastic work only by unremitting toil and ceaseless work. House surgeons should be encouraged to do plastic operations, but always with the assistance of or under the eye of the attending gynecologist. He who is compelled to teach himself does not always have the most proficient of instructors.

Finally, the operator must be born with, or must acquire, a "plastic imagination," if I may coin a phrase. The first examination, or, possibly, subsequent study of the patient, must unerringly disclose to him the nature of the operation which he will be called upon to do. Is the cervix merely eroded, or is it lacerated? Is there present simultaneously both a rectocele and a lacerated perineum, or do these exist separately? Do the symptoms and the findings justify the performance of an anterior colporrhaphy? These are questions which every man unconsciously answers for himself. But back of this reply, and dominating it completely, lies the controlling factor of all medical and surgical procedures—experience. It may be possible for one to learn how to perform plastic operations from reading; but no number of monographs or text-books can teach one judgment and experience.

The Technique of the Operation, Including After-treatment. Under this heading, the general statement must first be made that the main element leading to failure in plastic operations is a failure to appreciate the character of the operation to be performed. Küstner[3] has done good work in pointing out that typical or classical operations are not necessary when only a part of the vagina or perineum are involved. Thus, it is quite unnecessary to perform a typical Hegar or Emmett operation for rectocele or for a relaxed vaginal outlet if only the right sulcus or the left sulcus of the vagina is involved. His teaching goes even further in urging the operator to restore in his mind the exact contour of the original laceration, and to denude and suture this only. It is the ideal method, but like many ideal propositions, it works well only in isolated cases; but distinctly to be included in this group are the vaginal lacerations which lend themselves readily to this form of correction. The general statement may be left for discussion, that the operator shall not do more than to correct pathological conditions.

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[3] Kurzes Lehrbuch der Gynäkologie, Jena, 1901, p. 60, seq.

On the other hand, however, no factor contributes more frequently to failure than incompleteness. To attempt to correct a complete laceration of the perineum by the flap-splitting method, let us say, without denuding and repairing the invariable vaginal laceration, involves inevitable failure; and yet this is not an uncommon blunder. But perhaps the most common error in this category is the suture of the vaginal mucosa in either anterior or posterior colporrhapy without including the fascia in the former case, the muscles and fascia in the latter. And I must state, with all due deference to those who have strenuously advanced opinions to the contrary, that an operation for cystocele is not complete unless it includes a pushing back of the bladder and its retention in its new position by some method of suture. It is perfectly immaterial, I believe, what the form of the denudation is, provided the bladder is properly retained; but just as the Stolz operation of former days has been relegated to oblivion, so will all others be which depend merely on the principle of removing or denuding various figures or forms of the mucous membrane only of the anterior vaginal wall.

In precisely a similar way, operations involving the posterior wall will fail unless the fascia or muscles, or both—depending upon the variety or extent of laceration—are included in the suture. And again as to these operations, I think it is entirely immaterial what the figure of the denudation is, provided the original laceration is included and the suture is so directed as to embrace the torn underlying tissues.

One more word as to the general causes contributing to failure. The denudation or the suture of the underlying tissues must be so done that the suture of the mucous membrane will lie in the median line, or the resulting scar will deviate to one side or the other, marring the cosmetic effect of the operation. It is also important that like tissues be joined, avoiding the suture of mucosa to skin, fascia to mucosa, etc. This is such a self-evident proposition that I hesitate to mention it, but I have seen some ludicrous sequels resulting from this error.

Let me now briefly take up some of the minor details of operative technique which often lead to disaster. First and foremost is the preparation of the patient. Many an extensive and well-performed plastic operation has been ruined because the patient's bowels have moved during the procedure. This is because the last enema given did not come away clear. While the nurse is ostensibly at fault, the real fault lies in her insufficient understanding of the necessity for a perfectly empty rectum, and this is directly traceable to her instruction. Experienced and well-taught nurses do not make this blunder, and the fact that it occurs at all is an argument in favor of permanent head nurses, at least in gynecological wards. In line with the same mode of infection is the habit of some operators of inserting the finger in the rectum while suturing the posterior vaginal wall. While this is, at times, a safeguard against passing sutures too deeply, it almost goes without saying that it should not be done unless the finger is protected by a sterile finger-cot or glove; but the tying of sutures should then be done by an assistant.

A too liberal use of antiseptics, especially bichloride of mercury, and a too vigorous "scrubbing up" of the vagina, may so injure the vaginal mucous membrane that it will not heal readily. Plain sterile water contains all the necessary virtues for irrigating during the operation. The tissues attacked must also be subjected to as little bruising as possible; or, as in other parts of the body, healing will be decidedly interferred with.

As to sutures and suture materials, much could be written which is quite unnecessary. A very prominent cause of necrosis of tissue, following plastic operations, lies in tying the sutures too tightly, especially if they are of chromic catgut. Placing the sutures too near each other is another fruitful source of strangulation of the tissues, leading ultimately to healing by granulation. Personally, I believe it is not a matter of much moment what kind of suture material is used, provided the sutures are placed at proper intervals, are not tied too tightly, and, if they are of silk or silkworm-gut, are removed before they tear through, or strangulate the tissues, or become loose. Chromic catgut used as buried sutures, however, in posterior colporrhaphy, is to be avoided; as by its continued presence near the rectum it may become infected.

Perfect hemostasis is an important desideratum in plastic operations. I have seen a considerable number of cases in which secondary operations were necessary because the wound had been forced open by a hematoma from oozing vessels. Especially in cystocele operations the oozing is apt to be extensive if the plexus of veins lying between the uterus and the bladder is injured when the latter is pushed back. The arteries in the submucous tissues are usually small, but it is good technique to ligate them to avoid subsequent hematomas. For years Zweifel has been preaching perfect hemostasis in gynecology as a prophylactic measure against infection. In no department is it more important than in the one under consideration. A smal hematoma, while it may not tear asunder the sutures, is obviously a fertile source of infection.

A frequent source of functional failures in plastic operations lies in the too tight closure of the vulvar outlet. The anatomical result may be perfect, but if the denudation has been made too wide in the posterior wall, or too broad in the anterior wall, or both, coition and urination may be interferred with to a considerable extent. The reverse is also true, for even if the patient is functionally relieved or even cured of her symptoms, the result is a failure if the vulva gapes and the sulci still show the effects of the original laceration.

The common experience of finding only the skin of the perineum sutured and the muscles and fascia ununited has been touched upon. In no case of this kind can the functional result be worthy of consideration, since the anatomical lesion remains uncorrected.

In operations upon the cervix a not common, but still not rare, sequel is the finding of the cervical canal closed, with the development of a hematometra. I have seen this but twice; but it is, of course, due to imperfect technical procedure, and is an almost unpardonable blunder.

Turning now for a moment to the after-care of plastic cases, two points only will be mentioned. One is the blundering error of permitting a full-sized rectal tube to be passed in cases of complete laceration of the perineum. Unless a tube of small calibre is passed along the posterior rectal wall, infection with giving way of sutures is almost certain to occur, vitiating the entire operation. A suitable length of time, for the same reason, must be allowed to elapse before the bowels are permitted to be moved. These statements are so obvious that I would not mention them were it not that experience and observation have taught me that too often operations fail because of the omission of just such details. The second point is that catheterization must be rigidly aseptic; for a woman is not cured who leaves our hands with a chronic cystitis. This again is a subject for scrupulous teaching to nurses.

I am perfectly aware that I have omitted much that could be said about the causes of failure of plastic operations. I have tried to touch upon some of the essential sins of omission and commission. By no branch of their practice are gynecologists more closely judged than by their plastic work, and a thorough discussion of this subject cannot fail to bring out opinions which will be of general benefit.

CHRONIC EXTERNAL OPHTHALMOPLEGIA.

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SURGEON TO THE WILLS EYE HOSPITAL, AND PROFESSOR OF OPHTHALMOLOGY IN THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE.

The patient presenting a rather unusual form of disease, chronic external ophthalmoplegia, a girl sixteen years of age, who came to my service at the Wills Eye Hospital, said that she had always been well and strong, having been raised on a farm and having spent most of her time in the open air. Her mother died twelve years ago, of unknown cause, but her father is still alive and well. She never had brothers or sisters. The patient's grandmother had a goitre, but there were but few others with goitre in the region from which the patient came.

Examination showed the girl to be unusually large and strong for her age. The head was especially large and the features heavy, but there was no enlargement of the hands or feet. The thyroid was of normal size. Ptosis, and to a marked degree, existed upon both sides, the palpebral fissure on the right side being reduced to 5 mm., that of the left to 3 mm. in breadth (Fig. 1). Both eyes were fixed, the right eye looking straight ahead, with the left slightly divergent (Fig. 2), and all motion was abolished, except when the patient was requested to look down, when both eyes could be moved in that direction about 2 mm. Upon forceful efforts to raise the lids the palpebral fissures on both sides were made 2 mm. wider. The pupils were 3 mm. in size and were active to light and accommodation stimuli. In the right eye vision equalled $\frac{5}{9}$ and type 0.50 D. was read from 10 to 20 mm.; in the left vision equalled $\frac{5}{7}$, and type 0.50 D. was read from 10 to 20 mm.

Dr. Weisenberg kindly tested the muscles of the eyes and face for reactions of degeneration, but with negative results.

Chronic external ophthalmoplegia is a rare disease; it develops generally in infancy or early childhood, seldom later in life, without symptoms indicating involvement of other parts of the nervous system, and slowly progresses until all or nearly all of the extraocular muscles, including the levator, are palsied. The course is entirely chronic, and thirty years or more may elapse before the eyeballs are fixed and immobile. Both eyes are affected, though rarely simultaneously, symmetrical involvement of the muscles of both eyes being seldom observed. Diplopia is not often complained of, by reason of the slow development of the disease, and because of its appearance in early childhood, before binocular vision has been properly established. In many cases, however, double images can be evoked by suitable tests. Although the progress of the disease is subject to fluctuations, and there may be long periods during which the process seems to be at a standstill, after the maximum degree has been attained and the eyes are fixed, they remain so permanently. The pupils and the ciliary muscles escape in nearly all cases. Vision also is unaffected and the ophthalmoscope shows no fundus changes. The health is good and signs of involvement of other parts of the nervous system are absent. Only in exceptional cases is there a complaint of headache and marked drowsiness.

Although, as just stated, most of these cases arise in infancy, Wilbrand and Saenger do not think that the term "infantile nuclear atrophy," which was applied to them by Möbius is proper, as they assert that quite analogous cases, clinically, have been observed between the ages of fifteen and sixty years. Thus, of 32 cases collected by these authors, 6 developed in earliest childhood, 7 between two and six years of age, 5 between seven and twenty, 6 between twenty-one and thirty, 3 between thirty and fifty-three, while no record was made of the developmental period in 5 cases.

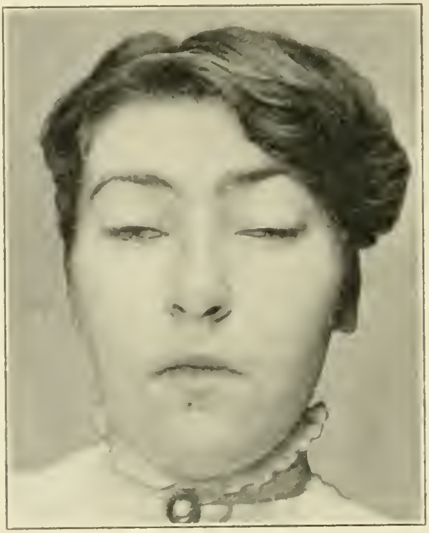

Fig. 1.—Chronic external ophthalmoplegia, showing pronounced ptosis, with fixation of both eyes in the primary position.

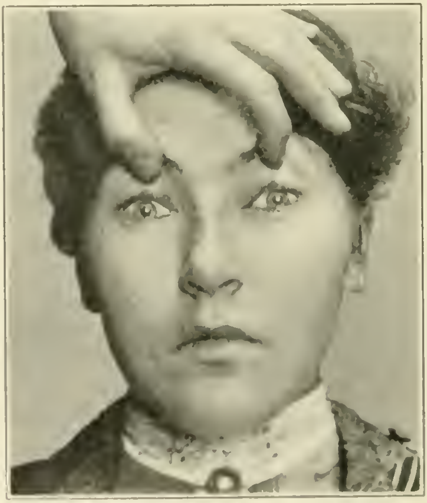

Fig. 2.—External ophthalmoplegia, showing fixation of both eyes—the left slightly divergent.

At its commencement the disease may assume a subacute character, gradually merging into a chronic progressive stage; usually, however, it is chronic from the beginning. Individual muscles are at first affected in some cases, this being particularly true of the levator, so that a unilateral or bilateral ptosis may be for some time the only ocular sign. Ptosis, however, is present in all cases, varying from a moderate drooping of the lid to complete closure. The degree of the ptosis also changes at different times, being increased usually by physical and psychic causes.

The diagnosis of the affection is not difficult, and, as suggested by Wilbrand and Saenger, it can usually be differentiated from other forms of ocular palsy by three points: (1) The binocular character of the extraocular muscle palsies, including the levator; (2) the absolute non-involvement of other parts of the nervous system, and (3) the frequency with which the disease begins in childhood.

It is often not easy, however, to differentiate the affection from congenital palsies, though in these cases the palsies are frequently unilateral, while in ophthalmoplegia externa they are always bilateral; congenital palsies are stationary, while those under discussion are progressive; and finally in congenital palsies there are frequently secondary deviations.

Nothing is known of the pathogenesis, as no uncomplicated case has come to section. A slow necrosis of the nervous elements, evidencing itself in an atrophic condition of the ganglion cells in the nuclei, with secondary atrophy of their nerve fibers, is the usual explanation of the condition. Although in many cases the nuclear degeneration which occasions the ocular palsies does not progress further, it is not unusual for the ophthalmoplegia to form part of a more general process and to be but the ocular manifestation of tabes or other diseases of the cerebrospinal system.

THE SECOND ANATOMICAL PROOF OF THE VALUE OF THE PARADOXICAL REFLEX.

By Alfred Gordon, M.D.,

ASSOCIATE IN NERVOUS AND MENTAL DISEASES, JEFFERSON MEDICAL COLLEGE, PHILADELPHIA; EXAMINER OF THE INSANE AT THE PHILADELPHIA GENERAL HOSPITAL.

At the February, 1906, meeting of the Philadelphia Neurological Society Dr. F. X. Dercum presented a communication on the occurrence of the paradoxical reflex in a case of localized hemorrhagic pachymeningitis.[1] He observed the presence of the reflex on the side opposite to the lesion before the operation and its disappearance after the clot was removed; then, again, its reappearance when a hemorrhage recurred. The latter was verified at autopsy. In this case the facility with which the reflex was elicited was striking, especially in contrast with the Babinski sign, which was not distinct. The persistence of this sign chiefly prompted Dr. Dereum to advise an operation. His idea proved to be correct.

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[1] Jour. Nerv. and Ment. Dis., September, 1906.

I now have the pleasure of submitting another case, in which my personal reasoning based mainly upon the existence of the paradoxical reflex proved also to be correct. It concerns a man, twenty-three years of age, sent to me by Dr. J. C. Da Costa at the Jefferson Hospital. He complained of pain in the right side of the head, in the same place where he received an injury six months prior: a heavy bar of iron fell on his head. Examination showed a cicatrix on the right side of the parietal region. Besides the headache the patient presented very pronounced mental hebetude and somnolence. The left knee-jerk was exaggerated and the paradoxical reflex was obtained with the greatest facility. There was no Babinski reflex, no Oppenheim reflex, no ankle clonus. Drs. Dercum and Mills kindly examined the patient and noted the striking contrast between the flexion of the toes on the right side and extension on the left, upon pressure on the calf muscles. They also verified the above statement concerning the absence of the Babinski and the Oppenheim reflexes. A trial of mercurials and iodides was given the patient, although there was no history of specific infection.

Dr. Da Costa trephined the skull at the level of the cicatrix and exposed the upper part of the Rolandic area. As the opening was made, the cerebral tissue bulged out; it was evidently under high tension. The opening was small and the surgeon immediately closed it up. Rapidly the patient regained his intelligence; the somnolence and the headache totally disappeared. As to the paradoxical reflex, it persisted during the first forty-eight hours; then gradually became weaker and weaker, so that at the end of the fourth day it could not be elicited. I saw the patient six weeks after the operation and found the paradoxical reflex, as well as the increase of the knee-jerk, totally absent.

Within the last few days another interesting observation came under my notice. A patient in Dr. W. W. Keen's service at the Jefferson Hospital was suffering from generalized epilepsy. I examined him first about a week prior to his admission and found all the reflexes normal. While in the hospital he had several seizures, which rendered him stuporous and confused. When an examination was made after the first attack I found increased knee-jerks, very slight Babinski, and a distinct paradoxical reflex on both sides; the latter were particularly marked and easily elicited. The abnormal reflexes persisted during the entire period of the attacks. Now it is the sixth day and there is no convulsion. The paradoxical reflex as well as others have totally disappeared.

From these two anatomical proofs I feel warranted in drawing the conclusions that I did from my clinical observations long before I had the good fortune to have operable cases. They are: The paradoxical reflex is a sign of value in the beginning of an organic affection of the motor system or in cases of its irritation. While the Babinski sign is of an inestimable value in cases with a definitely established lesion of the motor system, the paradoxical reflex, to say the least, is a sign of a transient irritation (as in the second anatomical case) or of a slight lesion of the same motor pathway.

REVIEWS.

Organic and Functional Nervous Diseases. By M. Allen Starr, M.D., Ph.D., LL.D., Sc.D., Professor of Neurology, College of Physicians and Surgeons, Columbia University, New York. Second edition. Pp. 816; 282 engravings and 26 plates in colors and monochrome. New York and Philadelphia: Lea Brothers and Co., 1907.

Upon its publication some four years ago, Dr. Starr's book immediately took front rank, being recognized as a work of superior merit by a distinguished clinician and investigator. Although at that time it well reflected the then existing state of our knowledge of diseases of the nervous system, the passage of time has added materially to this knowledge, changed and modified it in many respects, so that we find this second edition revised throughout, enlarged, modified in many places in conformity with advances in knowledge, and to a discussion of organic nervous diseases, of which only the first edition consisted, a section on functional nervous diseases has been added.

The major portion of the book, 712 pages, is devoted to organic nervous diseases; 86 pages only to the functional disorders. The section on organic nervous diseases is excellent throughout, many the chapters being of unusual merit. Of these one might mention especially those devoted to the diagnosis and location of diseases of the brain and the spinal cord, locomotor ataxia, tumors of the brain, the cranial nerves and their diseases, etc. The facts of significance in the diagnosis of focal and other diseases of the brain and the spinal cord are grouped together in two chapters that for orderly arrangement, terseness of statement, and lucidity of style cannot well be improved upon. Those familiar with Dr. Starr's pioneer work in the study and investigation of tumors of the brain will read with interest and satisfaction his chapter devoted to this subject. His discussion of other subjects is equally meritorious.

The section devoted to functional nervous disorders is as good as its rather cramped quarters permit; but it seems rather inappropriate to devote only 22 pages to epilepsy, 15 to neurasthenia and the occupation neuroses, 14 to hysteria, and 5 to migraine, while at the same time apportioning 61 pages to multiple neuritis. Although one would search in vain for the omission of any essential fact in connection with the discussion of functional nervous disorders, the widespread prevalence of neurasthenia, hysteria, and migraine, and their importance from a practical point of view, seem sufficient warrant for a relatively more detailed discussion of their symptomatology, diagnosis, and treatment, especially in the interest of the general practitioner. The reviewer also believes that the chapter on syphilis of the nervous system (3 pages) is somewhat inadequate—although this opinion probably results from his ideas regarding arrangement rather than from any lack of information in the book regarding the relation of syphilis to disease of the nervous system; but although the clinical phenomena of syphilis of the nervous system may not always suggest syphilis as the etiological factor, the protean manifestations of this infection suggest the wisdom of a rather detailed discussion of the subject in the interest of early diagnosis, and in the hope of effecting cure before irreparable damage has been done.

But these are details of minor importance. The book as a whole is excellent and reflects much credit on its distinguished author. As he states, "While the extensive literature of neurology has been carefully sifted, its facts collated, and its theories considered, the endeavor has been made to utilize personal observation and experience in the presentation of each subject." In this the author has well succeeded; the volume is a personal book, and reflects well the results of an analytical study of much clinical and pathological material. It may be cordially recommended to the specialist, as well as to the general practitioner, as a trustworthy guide, practical throughout, containing among other important matters many suggestions for treatment, including the results of recent advances in the surgical treatment of brain tumors, diseases of nerves, etc. A. K.

A Text-book of Diseases of Women. By J. Clarence Webster, B.A., M.D., Professor of Obstetrics and Gynecology in Rush Medical College, in affiliation with the University of Chicago, Chicago, Illinois. Pp. 712; 372 text illustrations, 10 colored plates. Philadelphia & London: W. B. Saunders Co., 1907.

The majority of prefaces might well be omitted, since, as a rule they are but so much waste space. Not so, however, the one introducing the present volume, in which Dr. Webster not only very definitely apologizes for presenting the book, but in which he also enters a very vigorous plea for a more wide appreciation of the extrapelvic portions of a woman's anatomy, when considering pelvic disorders.

The book opens with a most interesting and complete chapter upon the anatomy of the pelvis. Embryology, comparative anatomy, frozen sections, and ordinary gross dissections, each plays its part in making this the best chapter upon the subject in any textbook upon gynecology of which we have knowledge. In this, as in the succeeding chapters, by a system of large and small type, the author has distinguished between the more important material from a practical standpoint and that of less vital interest to the ordinary student, thus, we believe, performing a valuable service to his readers.

When considering the etiology of that form of dysmenorrhœa supposedly due to contraction of the cervix, he states his belief that the canal in these cases may be blocked by pieces of membrane, resulting in a partial damming up of the blood. In other words, he would widen greatly the class of membranous dysmenorrhœa, by including not only the cases usually classed here, but also those in which the extruded material is in small bits. Whether or not this is the correct explanation of these cases may be questioned, but it nevertheless is typical of the refreshing character of much of the volume.

We are glad to see the chapter upon the microörganismal flora of the genital tract, and we are much pleased with the saneness of his contribution upon neuroses in relation to pelvic disease. We wish that his advice in regard to the position of operative treatment in neurasthenia could be more generally appreciated, he believing that, while interference is proper if remediable lesions be present, on the other hand, operation is not to be placed in the fore-front of the therapeutic measures for the treatment of this condition, but that it is only to be invoked after careful neurological treatment has been carried out. A very interesting observation, which will be bitterly opposed in some quarters, is that in certain cases there is association between these neuroses and small cystic degeneration of the ovaries, and that therefore partial resection of these structures is to be recommended, upon the ground that there may be grave metabolic disturbance caused by the altered ovarian secretion.

We recommend the chapter upon case taking and physical examination to the careful attention of those especially concerned in the management of clinics. Not only is a valuable scheme given, similar to a number of others within our knowledge, but a most careful explanation of the different facts to be elucidated adds greatly to the value of the chapter. We note with pleasure that in his description of the various methods of cystoscopy he gives preference to that of Kelly.

Detailed consideration is given to the various minor gynecological measures, as the douche, tampon, etc., and in this connection the author strongly advises against the use of so-called pelvic massage, upon the ground not of its uselessness, since he admits that it has undoubted influence upon the blood and lymphatic currents and that therefore absorption of exudate may be produced, but because of its bad psychic influence upon the patient—an opinion with which we are in hearty accord, and which will be shared by the vast majority of competent men. As was to be expected the author, in dealing with the question on surgical technique, has written a chapter which is an embodyment of the best thought upon this important subject. He is, of course, an advocate of rubber gloves, and equally, of course, of their use dry. He does not believe that practical sterility can be obtained by the mechanical method alone, and he disapproves of bichloride of mercury in solution, because of the laboratory experiments of Leedom-Green and Opitz, who were able to culture staphylococci after appreciable exposure to this agent. He advocates the use of unpurified clove oil and seems very enthusiastic in reference to its virtues. We have never had any experience with this agent, but in common with a large number of others we are in a position to judge as to the practical value of bichloride of mercury, and we can only say that whatever may be the laboratory judgment, we believe that the results of numberless operations throughout the world have at least shown its practical utility. In this chapter upon technique the author has also included a very complete description of the vaginal method of attack, and he closes the chapter with an account of anesthesia, stating his belief in the employment of local agents whenever possible.

The consideration of the affections of the peritoneum is prefaced by a discussion of the etiology of acute general peritonitis in which is incorporated a considerable account of the experimental work which has been done upon this subject, thus rendering the chapter of particular value. Pelvic lacerations are to be repaired at the conclusion of labor, unless there are distinct contraindications, in which event a period of two months should be allowed to elapse before anything is done. We confess that we do not understand the rationale of this method. It has been well established that operative procedures to repair the injuries incident to labor are well borne, and that the results are perfectly satisfactory if performed a week after the completion of parturition; indeed, some operators are so wedded to this period that they will not repair these injuries immediately upon the completion of labor, but prefer to await the seventh day before doing any repair. In view of this fact we therefore cannot see any advantage in putting the patient back to bed for two or three weeks at a time when she has just about regained her health after the lying-in period. In old cases of complete laceration the author recommends the flap-splitting method. In this we cannot agree, as we believe that the older methods give as good if not better results. Be that as it may, we feel that the author has not given a description of the operation which he advocates, which is adequate for the ordinary student; nor are we pleased with the illustrations of this particular procedure. We are also disappointed in the single procedure recommended for the repair of cystocele. Not that it is not a method of value, but no mention has been made of other procedures which are needful in certain of the more marked cases of this injury. In considering the question of the pessary treatment in non-operable uterine prolapse we note the omission of the form designed by Menge. Certainly if the Zwanck and Hodge are noted the existence of the above-mentioned instrument should have been recognized.

In his chapter upon the diseases of the ovaries the author states that conservative operations have given him satisfaction in all but about 5 per cent. of his cases in recent years. He lays stress upon the accumulating evidence that removal of both ovaries has a decided effect upon the general metabolism of the patient. We are constrained to question whether the former statement will be consonant with the experience of the greater number of operators, but we are very glad that the author has emphasized the bad effects of castration, as we believe that even at the present day there is too great a tendency to look upon the ovaries as simple reproductive organs. We are also of the author's opinion that it is better, in a small percentage of cases, to have to reoperate than always to do a complete ovariotomy in doubtful cases, and while thus avoiding the later operation always to subject each patient to the grave changes which so frequently follow. Whether these changes are dependent or not upon the loss of some unknown ovarian influence makes no practical difference, as the effects are so often in evidence, whatever be the cause. We congratulate the author upon his presentation of the gross cystic diseases of the ovaries with which this chapter closes.

The consideration of the diseased conditions of the Fallopian tubes is fully up to the standard set by the rest of the book, but we do not think that the illustration depicting the evacuation of an abscess in the cul-de-sac is well executed, as the direction of the incision is too far posterior and away from the cervix. In the chapter dealing with metritis of an acute variety in connection with labor or abortion, we feel that it would have been of advantage had the value of the examination of the blood in obscure cases been insisted upon as an aid to diagnosis, and also that in describing the method of intra-uterine culture there should have been an explanation of the limitations of the method. We are delighted at the clearness of the teaching in regard to the dangers of the curette and the slight value of intra-uterine douches in these cases, while, on the other hand, the importance of ergot and the value of intra-uterine application of formalin is well emphasized. We feel it to be possible that the future may show that the author has been a little too skeptical with regard to the benefit to be gained from the use of the antistreptococcic serum.

We are particularly pleased with the chapter dealing with the displacements of the uterus; it is thorough and due credit is given the various operations which have been devised. To meet the indications for ventrosuspension or the Alexander operation the author advises that the round ligaments be utilized, anchoring them to the posterior uterine surface without cutting them. He would more stringently limit the indications for the Alexander operation than is usual among those operators who depend upon the round ligaments for operative correction; we believe this to be a wise attitude. We would call particular attention to the chapter of fifty pages upon the fibromyomas of the uterus. For this chapter there can be nothing but praise.

Our only criticism of the chapter upon extra-uterine pregnancy is that the treatment of advanced cases has been slighted, being too fragmentary and uncertain in tone to be of any great value. While, of course, the rarity of this condition makes this omission of little actual importance, it seems that a more definite teaching would be in place if the subject is to be mentioned at all.

Several extremely interesting pages are devoted to the appendix in relation to pelvic disease. The author does not admit the existence of the ligament of Claudo, for which we thank him, and he believes that while the appendix may be diseased at the same time with, but independently of, the pelvic structures, its pathological condition may be either consequent to or a cause of pelvic disease. This pathological relationship is occasioned by contiguity and not by lymphatic transmisson. The scientific scope of the whole book is well typified by the consideration of the white cells in their relation to infectious processes, and embodies the best recent work upon the subject. Enteroptosis is considered in a very conservative and satisfactory manner, but we think that more space should have been given to the operative treatment for the condition. The last chapter, that upon sterility in the female, begins with a statement which we confess we do not understand. The illustrations are, as a rule, of marked excellence, though some are marred by a too diagrammatic tendency, and we notice with pleasure that, although they are ample for all needful elucidation, there is a grateful departure from certain recent gynecological picture books.

As a whole the volume is one of the most praiseworthy of which we have any knowledge. It does not claim to be a purely operative manual, and yet with the few exceptions which we have noted it gives ample space to the description of the methods which the author has proved in his own work. Moreover it is well written, and is therefore very readable—a desideratum not always attained in medical works. Finally it is a gynecology in the strict sense of the term, treating diseases only which are peculiar to women; it does not attempt to enter the domain of general surgery by describing operative procedures, as nephropexy, appendectomy etc., which, often demanded as they are in conjunction with pelvic operations, cannot, strictly speaking, be considered as within its scope. We believe that this limitation, which in some quarters may be considered as an omission, is praiseworthy, as we are still of the opinion that gynecology is worthy of a place among the specialties and that there have been too many attempts to fuse it with general abdominal surgery. W. R. N.

CLINICAL DIAGNOSIS. A TEXT-BOOK OF CLINICAL MICROSCOPY AND CLINICAL CHEMISTRY FOR MEDICAL STUDENTS, LABORATORY WORKERS, AND PRACTITIONERS OF MEDICINE. By CHARLES PHILLIPS EMERSON, A.B., M.D., Associate in Medicine, Johns Hopkins University, Baltimore, Maryland, with an introduction by WM. OSLER, M.D. Pp. 641; 126 illustrations; 6 colored plates. Philadelphia and London: J. B. Lippincott & Co.

THE essential function of a book on this subject is to provide a complete and absolutely accurate description of the methods that are useful to the clinician and the clinical laboratory worker, and to give a critical discussion of the relative value of each method and of the value or lack of value of the results so far as they bear upon diagnosis. It was to be expected that no one in this country would produce a more satisfactory book of this kind than the author of this one, because of his extensive experience in teaching these subjects and his wide training in various lines bearing upon such matters. In some ways this expectation has been realized; in others, it has not.

The chief failure in the book is, unfortunately, the most important one, namely: there are many inaccuracies in it; and, there being many such, it must be very difficult for anyone using the book to be able to determine upon which methods he can rely, as they are here given, and upon which he cannot rely. The obscurities and errors are at times such that it is impossible to believe that the author himself would deliberately stand sponser for them; and they must have slipped in accidentally, in proof-reading or in some other manner, in the process of making the book. Some of them are comparatively trivial; as, for instance, the statement that poikilocytosis is a change in the size or shape of the red blood corpuscles. Others of them are much more important, as they may readily lead to serious errors or omissions in carrying out methods. This is particularly true of a number of the chemical methods described. For example, it tells one nothing to say simply that a safety tube is used in Folin's method for urea when it is really a special safety tube. Further, Folin directs that the solution be not allowed to remain alkaline, while the description in this book says it must remain alkaline; again, the book tells one to continue heating for a half hour, while Folin stated, five years ago, that this is not long enough; and what does it mean to say that it is distilled until the ammonia is "almost all over?"

Except for this disadvantage, which, however, is sufficiently important to deserve the emphasis that has been placed upon it, the book is, in most ways, very satisfactory; and in some essential things, extremely so. It suffers from a somewhat involved and obscure style of stating facts, but it has the very marked advantage of evincing a close acquaintance with the large number of methods in use, a critical sense of the value or lack of value of the various methods, and a critical conception, likewise, of the importance or lack of importance of the results to the clinician. The latter point is illustrated in the discussion of the tests of the functional capacity of the kidneys. R. L. C.

THE COMMON BACTERIAL INFECTIONS OF THE DIGESTIVE TRACT. By C. A. HERTER, M.D., Professor of Pharmacology and Therapeutics in Columbia University, New York. Pp. 360. New York: The Macmillan Company, 1907.

WHEN we realize that bacteria form a fifth to a third part of human feces by actual weight, we appreciate the important part that microörganisms must play in the intestine. Unfortunately former observers have been altogether too ready to assume that this bacterial activity must be deleterious to the organism, whereas later investigations tend to show that a certain number of bacteria are necessary for the normal functions of the bowel. This mistaken conception of the significance of the intestinal microörganisms has caused a cloud of false hypotheses to arise about the subject of intestinal auto-intoxication, giving it a very undue prominence in the causation of disease. Most obscure conditions at one time or another have been ascribed to auto-intoxication without any scientific basis. The best way to set right such a mass of mistaken ideas is not only by ruthlessly trimming away wrong conceptions, but by giving us in their stead the real facts and showing how much truth there may be in auto-intoxication as a cause of disease, even though the whole subject is not yet capable of mathematical demonstration. This is what Dr. Herter does in the volume under consideration. The title "Bacterial Infections" is not a misnomer, but rather an understatement of the scope of the book. He not only considers infections, but also the whole subject of the relations which the intestinal flora has been shown to bear to digestion, to auto-intoxication, and to infection. The methods of investigations are very completely given, and there are chapters upon the comparative flora of herbivora and carnivora. Clinical sections upon the types of chronic excessive intestinal putrefaction and the methods relating to its control complete the volume.

The author is peculiarly fitted to write such a work, since he is at once an investigator, a clinician, and a teacher of therapeutics. The result of his labor is to be recommended to everyone who wishes to understand all that we know of the bacterial processes in the intestine, and surely no branch of pathological physiology is more important. J. D. S.

A Manual of Normal Histology and Organography. By Charles Hill, Ph.D., M.D., Assistant Professor of Histology and Embryology in the Northwestern University Medical School, Chicago. Pp. 463. Philadelphia and London: W. B. Saunders Co., 1906.

The value of even the best manuals of histology, of pathology, or of similar subjects is, of necessity, limited; and their usefulness to a student must depend upon the accuracy and vividness of the illustrations and the lucidity of the text. Of a manual of histology this is eminently true, for the author is confined, in a general way, to the objective description of the microscopic appearance of cells, tissues, and organs. Dr. Hill's arrangement of his book is much the same as is followed in other short manuals of histology, but he develops his chapters in a jerky and confusing manner, referring now and again to surgery, therapeutics, pathology, physiology, and clinical medicine. These references are not only confusing and ill-timed, but frequently inaccurate. Even to refer to the physiology of the liver by the statement that "The function of these cells [liver cells] is twofold: (1) to secrete bile into the bile capillaries; and (2) receive and contribute sugar to the blood capillaries," is unfortunate.

We regret to say that the illustrations aid but little. Those which are reprinted from other books are often well chosen, but many of the original cuts are inexcusably poor, while some convey no impression whatsoever. The most useful part of the book is the section upon the teeth. This is developed in greater detail than any other subject, and covers half of the chapter on the digestive system.

W. T. L.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Origin of Renal Hemorrhages in Nephritis.—KUSUMATO (*Arch. f. klin. Med.*, 1907, lxxxix, 405). Askinazy has laid stress upon the not infrequent occurrence of hemorrhage in cases of chronic nephritis. In several of the cases postmortem examination showed that the bleeding came from the renal pelves. Kusumato reports a similar case. A man, aged fifty-five years, presenting a typical clinical picture of chronic interstitial nephritis had on a number of occasions quite marked hematuria. The first bleeding occurred in conjunction with a transient hemiplegia coming on while straining at stool and the last with a fatal apoplectic attack. Careful microscopic examination of the renal pelves showed three extensive submucous blood extravasations which could be definitely traced to small ruptured veins. The accompanying illustrations are quite conclusive, and the point is of importance, as such hemorrhages are usually ascribed to diapedesis. Israel has suggested that the bleeding may be due to a sudden arterial hyperemia, basing his view upon observations made during operations. Kusumato points out that during unusual exertion, such as straining at stool, there is increased arterial tension and at the same time venous congestion, both factors probably being of importance in causing the rupture of the veins.

Paroxysmal Tachycardia.—E. SCHMOLL (*Arch. f. klin. Med.*, 1907, lxxxix, 594). There are at present two views regarding the nature of tachycardia. Hoffman has called attention to the frequent occurrence of a doubling or quadrupling of the previous pulse rate and assumes that the impulses at their point of origin are sent out much more rapidly than the heart is normally able to respond. Owing to some change in irritability during the attacks of tachycardia two or even four times as many impulses may pass through. According to Mackenzie the condition depends upon a heaping of extra systoles. Schmoll reports two interesting cases with careful analysis of the pulse tracings. In the first case the normal auricular impulse is shown on the venous tracing, but during an attack is replaced by a ventricular venous pulse. Tracings taken during intervals give an explanation of this phenomenon. Certain irregularities can be interpreted only as extra systoles and the absense of an auricular impulse before these contractions mark them as ventricular extra systoles. The disturbance of rhythm, the absence of the compensatory pause, and the occasional occurrence of atrioventricular extra systoles with shortened auriculoventricular pause indicate the bundle of His as their point of origin. In the second case during attacks there was often a typical pulsus alternans, an alternating strong and weak contraction of the auricle as well as of the ventricle and the two occurring independently. The interpretation of the tracings is rendered difficult by the occurrence of abortive extra systoles. A slight wave of the auricular contraction is recorded without a succeeding ventricular wave, but auscultation shows that a contraction does occur. As in the first case these extra systoles are not followed by a compensatory pause and the time of transmission of the impulse from the auricle to the ventricle is shortened. Here then are two cases of tachycardia depending upon the occurrence of extra systoles which probably arise in the bundle of His

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Sodium Chloride Metabolism.—Bittorf and Jochman (*Arch. f. klin. Med.*, 1907, lxxxix, 485) have studied the metabolism of sodium chloride and its relation to water excretion in a large number of pathological conditions. Considering the importance which has been laid upon sodium chloride retention as a cause of œdema their contribution is of considerable value. Among the infectious diseases they have investigated lobar pneumonia and find that the chloride retention is due not to a primary retention of water, but to a marked affinity of the pneumonic exudate for salt, the organism being able to react during increasing consolidation by an increased elimination of water, while the salt excretion is reduced. They were unable to influence the chloride output by the administration of 15 grams of salt. In cases of acute exudative inflammation, such as pleurisy, the chloride output may be normal and the administration of large amounts of salt need not lead to a chloride retention. There is not even a delayed excretion of the salt, and indeed it may exercise a diuretic action and stimulate a further chloride excretion. After exploratory puncture there may be an increased sodium chloride excretion due to absorption from the exudate. Diuretin increases the water output but has no influence upon the salt.

In heart cases and in cases of chronic nephritis complicated with broken cardiac compensation the elimination of water and salt depends entirely upon the circulatory condition. The two substances bear no definite relation to one another and each may be influenced quite independently of the other. Even in the presence of œdema a considerable intake of salt need not lead to water retention; indeed, in some instances it acts as an efficient diuretic.

In nephritis, conditions are found to vary. In an instance of acute nephritis with œdema administration of salt led to increased salt and water excretion, while at a later period when the œdema had cleared up it was followed by profuse water elimination but retarded salt elimination. In chronic parenchymatous nephritis the same patient may show at intervals very good, good, or delayed salt excretion with normal water elimination. In one instance sodium chloride administration displayed a marked diuretic action carrying with it an excess of salt. As a rule, in chronic interstitial nephritis there is also good capacity for sodium chloride elimination. In some instances there was good salt elimination with poor accommodation for increased water output. In other instances both were deficient. The chloride content of non-nephritic exudates, transudates, and œdemas is often higher than those of nephritis.

From these observations Bittorf and Jochman conclude that one is unable to judge of the severity or character of a renal lesion by the capacity for eliminating chlorides; that salt retention is not the primary cause of œdema, changes in the bloodvessels being a far more important factor; that sodium chloride may be administered without any harmful consequences; and that the modern demand for a salt-free diet based upon the assumption of a diminished capacity for salt elimination is without scientific foundation.

The Action of Dechloridation upon Bradycardia and its Interpretation According to the Myogenic Theory of the Heart Beat.—ENRIQUEZ and AMBARD (*La semaine méd.*, 1907, No. 4, 40) have been led by the reports in the literature of the beneficial effects of the deprivation of salt in bradycardia to try it in a case of their own and had very good results. In one of the reported cases the beneficial action was noted most markedly on a milk diet, the rate becoming normal, but as soon as a mixed diet was resumed the rhythm became slow again. Their patient was a woman, aged fifty-seven years, who complained of pains in the knees, dyspnœa, and dizziness. The pulse varied at first between 32 and 39 to the minute, the blood pressure was about 230 mm. of mercury. The urine contained no albumin. The patient was put on a salt-free diet and eight days afterward the pulse was 42, the dyspnœa and dizziness diminishing. At the end of seven weeks on the diet the pulse became normal (72) and the shocks of the two sounds were normal in rate and rhythm. The symptoms all ceased and the patient gained about fifteen pounds in weight. During the treatment the woman was able to do her ordinary work. Enriquez and Ambard discuss briefly the two theories as regards the heart beat (the neurogenic and the myogenic) and give brief abstracts of most of the recent work done, especially in proof of the myogenic theory and the functions of the bundle of His. The recent work on the action of solutions of salt on tissue cells is reviewed: their toxicity, aside from the effects of osmosis, seems to be directly injurious to the musculature and independent of the innervation. The results of deprivation of salt in bradycardia seems to bear out these conceptions. It is rather interesting that the heart in this case passed through the different stages of arrhythmia in slowly increasing to the normal rate which the heart in Erlanger's experiments on compression of the bundle of His showed, but in the reverse order. In conclusion they say "The effect of dechloridation upon slow pulse agrees perfectly with the myogenic theory, and the analysis of the pathogenic condition, the slow pulse, shows us a curious and hitherto unique fact of the injurious action of chlorides upon a diseased muscle without any intervention of the nervous system."

Stokes-Adams' Disease due to a Sclerogummatous Lesion of the Bundle of His.—Vaquez and Esmein (*La presse méd.*, 1907, No. 8, 57) first give a detailed account of the physiology of the heart with many references to the literature, as regards the neurogenic and myogenic theory of the heart beat, and quote from the more recent work on heart block and arrhythmias in proof of the myogenic theory. This careful review is divided into the anatomical description of the bundle of His by various authors; the experimental proofs of its relation to the heart beat by sectioning it completely and partially, and noting its effects on the pulse; and the clinical evidence in the recently reported cases of arrhythmia, bradycardia, and Stokes-Adams' disease. The following case is reported by Vaquez and Esmein as of special interest, as it was followed carefully through life with excellent pulse tracings and cardiograms, and then carefully examined at autopsy. The patient, aged forty-three years, a few months previously began to have attacks of syncope with convulsions, these gradually increasing in frequency to date of first observation. He gave a history of syphilis and had a double aortic lesion. The pulse varied from 50 to 60 to the minute, but in the syncopal attacks was as low as 30 to 60 to the minute. The attacks become very severe with convulsions and opisthotonos, and at times the pause in the pulse lasted as long as six seconds. Medication had little or no effect and the patient died in an attack after about four months of observation. On careful examination the difference between the arterial and the venous rhythm was quite marked, the jugular pulsations being well seen during the pause in the pulse. On auscultation during the pause the contractions at the base of the heart could be faintly heard. At autopsy the heart showed a double aortic lesion. Its weight was about 370 grams. The nervous system, pneumogastrics and sympathetic nerves were normal. On examining the heart a plaque of sclerosis, irregular in outline and about 2 x 3 cm. was found lying between the auricles and ventricle, just in the region of the bundle of His. Microscopically, the fibers in the bundle were found to be surrounded and compressed by the sclerotic tissue, thus giving rise by their pressure to the irregularity in the pulse and the manifestations of heart block.

An Extraordinary Blood The Presence of Atypical Phagocytic Cells.—F. Van Nuys (*Boston Med. and Surg. Jour.*, 1907, 156, 390) reports the history of a man aged fifty-five years whose complaint was of swollen feet and passage of a tape-worm. His family and personal history were unimportant For six months the feet had been swollen and lately there had been some dyspnœa and weakness. Examination revealed a bronchitis and a fairly compensated aortic regurgitation. The liver and spleen were quite large, the former reaching three inches below the costal margin and the latter nearly to the umbilicus. In the region of the descending colon were two egg-shaped hard masses which did not wholly disappear after the bowels were thoroughly moved. There was some œdema of the feet and bases of the lungs. On treatment a beef tape-worm was passed some hours after admission. No blood was found in the stools. Eggs of Tricocephalus dispar were always present. Urine and sputum were negative.

The blood was examined on account of the peculiar features, and control counts were made from both ears and fingers. Daily and weekly control differential counts were also made. The number of leukocytes varied greatly from time to time. During the stay in the hospital the patient had a short attack of erysipelas after which there was a fall in the number of leukocytes to below normal. He left the hospital in about the same condition as on admission and died a few hours afterward. No autopsy was obtainable.

The characteristic features of the blood were the presence of large basophilic phagocytic cells capable of amœboid movements and very different from any previous cells described in the blood. (These are shown very well in the plates.) There were two varieties of these cells: first, large mononuclears with granular and vacuolated cytoplasm and with large well-stained, oval nuclei. These cells occur in plaques resembling endothelium. They were actively phagocytic and had a chemiotactic influence on the leukocytes. Very rarely red cells or lymphocytes were enclosed. Their average size was 23 x 16.9 μ. The other type was a large multinuclear phagocytic giant cell with long fantastic streamers of protoplasm. The nuclei were the same as in the other variety. The cell membrane was hard to determine. Between these extremes there were many cells having the characteristics of both. These giant cells were apparently long-lived and showed leukocytes in various stages of decomposition inside them. These abnormal cells varied enormously in numbers from day to day, at one time being being about 40 per cent. in a count of 1000 cells. After the erysipelas they almost disappeared but began to recur shortly before death.

In classifying them as basophilic they come under the same heading as lymphocytes and transitional cells. At no time were eosinophiles, mast cells, or myelocytes seen. Normoblasts were rare. There was no polychromatophilia and only slight poikilocytosis and a moderate achromia of the red cells. The specific gravity of the blood was 1044; the coagulation time about five and a half minutes. Blood platelets were twice counted; once they were 391,700 per c.mm. and once 276,000. The red cells varied from 2,500,000 to 4,000,000. The whites were very irregular in numbers, reaching as high as 100,000 and as low as 5000. Experiments tended to show that there was an absence of digestive ferment in the cells, such as myelocytes are said to possess in myelogenous leukemia; hence it is probable that these cells did not come from a tumor of the bone marrow. There was no albumose in the urine. Van Nuys concludes that the morphology and plaque formation suggest that they are endothelial in origin. Undoubtedly, somewhere in the man's body a neoplasm was setting loose these cells into the blood. If the phagocytes arrived in the blood stream by way of the lymph channels we might expect the inclusions to be lymphocytes. The enlarged spleen and the mysterious abdominal lumps possibly held the secret. The case might be considered leukemia, meaning by this a malignant disease of the blood.

A study of an additional case in the wards at present with a similar blood picture will be reported later and may help to clear up this peculiar condition.

Cor Triloculare Biatrium.—J. D. Mann (*Brit. Med. Jour.*, 1907, i, 614) reports on this unique case of three-chambered heart in a man aged thirty-five years, who appeared to have some chronic heart disease with the usual symptoms. He was dyspnœic, but not cyanosed, nor were the fingers clubbed. The area of cardiac dulness was increased and the cardiac impulse more extensive than usual. There was no thrill, but there was a loud, rough, systolic murmur over the whole cardiac area. Later, before death, the patient became very cyanotic, the heart was tumultuous and irregular, and there was general œdema. At autopsy there was found to be a complete absence of the septum between the ventricles, and the walls were of equal thickness. Into the common ventricle opened the mitral and tricuspid orifices in their normal positions. Two arterial trunks emerged from the ventricle in the position of the aorta and pulmonary artery; the valves being somewhat sclerosed; the auricles were normal excepting for a patent foramen ovale. The most interesting point is the age of the patient as these cases are extremely rare even in childhood, and this was a man who was little inconvenienced by his cardiac condition until his last illness. As a youth he was rarely sick and only at times dyspnœic. He was never cyanosed until the last three weeks of his life.

Mann then gives brief summaries of cases in the literature reaching adult life, and discusses the question of the circulation in such cases and the cause of congenital cyanosis and polycythemia.

In conclusion, he says that the conditions which give rise to the cyanosis in cardiac malformations remain obscure. In addition, however, to the results that are directly due to the action of the malformed heart on the blood current (mixture of venous and arterial blood), it appears probable that a permanently dilated vascular system shares in the production of the cyanosis.

SURGERY.

UNDER THE CHARGE OF

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The Employment of the X-rays for Locating the Position of the Accessory Sinuses of the Nose and the Diagnosis of their Diseases. Goldmann and Killian (*Beit. z. klin. Chir.*, 1907, xliv, 1)) say that the outspoken view of Scheier in 1897, concerning the value of the x-rays in the domain of rhinolaryngology, has been rapidly established. The accessory cavities of the nose have given particular trouble. In the earlier efforts the lateral exposure of the skull to the rays was almost exclusively employed, because little faith was placed in the anteroposterior exposure. Goldmann and Killian had their attention first attracted to the value of the lateral exposure by the opportunity of observing the excellent results obtained by Professor Hagler in Basel, who secured very clear skiagrams of the frontal and maxillary sinuses. Their first efforts were directed toward showing the normal frontal sinuses, from which they became convinced that the lateral exposure gave the best obtainable result. By this method it could be decided with certainty whether or not the frontal sinus was present. This was proved in a case in which probing could not detect the presence of the sinus. The *x*-ray plate showed practically no sinus, which was confirmed later by operation. With much clearness it can be shown whether the sinuses are equally or unequally developed, or whether a septum completely divides the two sides. It shows also how its different loculi are shaped and the size of the sinus. These facts are of much value in connection with operations on the sinus. A lateral exposure would not determine the side involved, since both cavities would then lie over each other. An anteroposterior view would permit a comparison between the affected and the non-affected sides of the frontal, ethmoidal, and maxillary sinuses. A purulent collection on one side can thus be recognized, since that side would be considerably darker than the other. One side may be slightly obscured from a swollen, degenerated mucous membrane or to a less extent from an œdematous mucosa. Only a deep shadow permits a positive diagnosis. When both sides are darkened the interpretation is more difficult, although one side may show a deeper shadow than the other. While the presence of a marked obscurity on both sides permits the diagnosis of double-sided disease, the absence of such obscurity does not positively exclude disease on both sides. In the cases reported almost without exception a darkening on one side in the region of the ethmoidal cells proved at operation to be due to disease in these cells. While Goldmann and Killian strongly prefer exposure in the sagittal plane they do not reject the lateral exposure entirely, but employ it sometimes to supplement the other. They claim that the anteroposterior exposure to the rays is an especially valuable method of establishing the topographico-anatomical relations of the accessory sinuses and their diseases. It should be classed as an equivalent with the other methods of examination, serving to substantiate their findings and making them not indispensable.

The Treatment of Gangrenous Herniæ.—Hesse (*Beit. z. klin. Chir.*, 1907, xliv, 172) says that the treatment of gangrenous strangulated herniæ has varied for the past thirty years, chiefly between the making of an artificial anus and primary resection with reunion of the divided ends. Mickulicz and Kocher favor primary resection; Bramann favors the artificial anus. Hesse says that the review of a large mass of statistical material gives convincing evidence that resection is recognized as the best treatment for gangrenous herniæ. There is much diversity of opinion as to the indications for the operation. Lotheissen, for example, prefers an artificial anus if there is the least sign of peritonitis; Barker likewise in old and desperate cases; others consider advancing decay as a contra-indication. Of 197 cases of incarcerated hernia coming to operation in the Tübingen Clinic, in 56 there was a suspicion of gangrene, or 23.34 per cent. Of these 56 primary resection was performed on 40 cases, with a mortality of 47.5 per cent; and on 3 the operation for artificial anus was done, with a mortality of 100 per cent. Of the 56 patients, 33, or 58.9 per cent., died. Peterson's results in the Heidelberg Clinic, from 1901 to 1905, showed 8 deaths in 24 cases of primary resection and 7 deaths in 8 cases of artificial anus. In a total of 860 cases of primary resection for gangrenous hernia collected from the literature 382 died, a mortality of 44 per cent., and of 604 cases in which an artificial anus was made, 431 died, a mortality of 71.3 per cent.

With regard to the question as to the relative value of local and general anesthesia, Hesse says that as a rule the local is to be preferred, although it has a more serious influence on the mental condition than general anesthesia. The danger of shock with local anesthesia is considerable, and the psychical influence interferes with heart action quite as much as chloroform or ether. Hesse concludes from his extensive study that there is only one rational treatment for gangrenous hernia, the complete resection of the gangrenous portion of the intestine, while the artificial anus as a life-saving operation should no longer be given consideration.

The Etiology of Lateral Abdominal Herniæ.—Blauel (*Beit. z. klin. Chir.*, 1907, xliv, 229) defines these herniæ, according to Graser, as those coming through the abdominal wall in the linea semicircularis or the oblique muscles, or the tendinous expansions of the latter. This definition excludes those coming through posterior to the oblique muscles, the herniæ of the loin. Our knowledge of the causation of these herniæ has received important contributions from Quervain, Borchadt, and v. Baracz, each of whom reported a case. Before the classic work of Schmidt, only wounds and suppurative conditions were recognized as causes. Then Wyss showed that hereditary defects of the muscles were responsible for congenital cases, which permitted the above authors to show that muscular atrophy could be a cause. In such cases there is a circumscribed ectasia of the abdominal wall, but no essential hernial sac or ring. Borchadt called them pseudoherniæ. Baracz and Blauel accept this term, which should be reserved for those cases resulting from an atrophy of the muscles. Baracz attribued the atrophy to a spinal disease and its associated paralysis. Quervain discussed the etiology exhaustively and concluded that only the influence of the nervous system would explain it. In the case which he reported the atrophic region was supplied by the anterior branch of the twelfth intercostal nerve. In the most recent text-books of surgery the question of the causation of these herniæ from muscular atrophy is left open. Blauel reports a fourth case in which the symptoms of pseudohernia were the same as in the other three, already referred to. In recumbency and easy respiration nothing especially protruded. With intra-abdominal pressure a localized, semiglobular tumor protruded, at the edges of which could be felt a well-defined deficiency in the abdominal wall. With disappearance of the intra-abdominal pressure and the tumor, the edges of the deficiency in the abdominal muscles were soft and yielding. Electricity showed a degenerative atrophy of the muscles on the right side of the abdomen. The cause in this case was definitely shown to be an anterior acute poliomyelitis, of which there was marked evidence in the paralysis of the right lower extremity. This is the first case in surgical literature in which this cause has been definitely shown to be present, although four cases have been reported in the neurological literature by Ibrahain and Hermann. Another case has been reported of isolated partial paralysis of the abdominal muscles from this cause, without, however, the signs of a lateral abdominal hernia. Blauel believes that acute anterior poliomyelitis is the usual cause of lateral abdominal pseudoherniæ.

A Study of Some Points Concerning Urinary Troubles in Retroversion of the Gravid Uterus.—Trillat (*Annales des maladies des organes génito-urinaires*, 1907, xxv, 561) says that he desires to call attention to the mechanism of retention of urine, the explanation of the correction of retroversion by the simple evacuating catheter, and the particular significance of Laroyenne's symptom, micturition from feeble pressure upon the posterior inferior part of the vagina. He found by his investigations that the uterine neck was 7 cm. above the upper border of the symphysis, which he considers an important point in cases of great distention of the bladder. The urethra attains a length of 8 to 9 cm., nearly three times the normal. From the urethra to the upper end of the cervix uteri measured 7 cm., the bladder in this portion forming what he calls the vesical infundibulum. The cervix pushing forward into the bladder increases the length of the urethra by the formation of this infundibulum. While complete incarceration of the uterus in the pelvis gives severe vesical conditions, a partial incarceration can also give grave phenomena. The retention of urine has been accounted for in various ways by different authorities, such as curvation of the urethra, diminution of the calibre by elongation, œdema of the uterine neck, paralysis of the vesical muscle, and direct pressure of the cervix on the urethra and neck of the bladder. Trillat believes that the obstruction is due to the pressure forward of the cervix from the retroversion against the lower part of the bladder, the infundibulum, occluding its cavity. If light pressure is made with the finger in the posterior vault of the vagina against the retroverted uterus urine will escape from the urethra. If the pressure is removed the escape of urine will cease. Trillat says that the establishment of this symptom alone should make one think of retroversion of the gravid uterus, and thus a dangerous error and its consequences be avoided. Laroyenne believed that the finger pressed on a prolongation of the bladder behind the uterus. Trillat says that it is impossible to admit this with a gravid uterus after the fourth month, when the troubles begin, since there would be no room for such a prolongation. The escape of urine he thinks, is due to the removal of the obstacle to the outflow of urine, produced by pushing the body of the uterus forward, thus relieving the pressure of the cervix against the lower part of the bladder or infundibulum.

A Case of Pressure on the Trachea.—Hall (*Lancet*, 1907, i, 1079) reports such a case, which at first had been treated for symptoms of influenza. This cleared up in a week sufficiently to permit the patient to go to work. A fortnight later he was treated again, this time for asthma. Two months after the beginning of the trouble he had to be propped up in bed; he was somewhat cyanosed, with a pulse of 150 and very feeble. Examination of the larynx and chest showed little evidence of air entering or leaving the chest, evidently the result of some obstacle in the respiratory passages. The laryngoscopic examination excluded obstruction of the glottis, and the absence of amelioration from time to time excluded spasm of the smaller bronchi as in asthma. As both lungs were equally affected, the probability was in favor of tracheal stenosis. At the necropsy a mass of enlarged glands was found compressing the trachea, just at its bifurcation. A microscopic examination showed it to be lymphosarcomatous. The rapidity with which the compression of the trachea developed was very interesting. In the beginning of January, though weak from his recent attack of influenza, he was otherwise well, and yet on the following February 19, he was found to be suffering from severe dyspnœa.

Observations on the Operative Treatment of Muscular Wryneck.—Böcker (*Zentralbl. f. Chir.*, 1907, xxxiv, 449) reports the results of the operative treatment of torticollis in Hoffa's clinic. Of the many methods of operative treatment proposed, only two, in his opinion, deserve to be considered: the simple tenotomy of the sternomastoid of Volkman and the partial resection of Mickuliez. Doering reproted 35 cases with good results in all except 1, from division of the stretched fibers of the cucullaris with abundant overcorrection, and considers this the proper treatment for all cases, mild and severe. Böcker reports good final results from this method in mild and early cases. He reports that since 1898, 120 cases have been operated on, 90 by the Mickuliez and 30 by the Volkman method. When the final results are compared the Mickuliez operation is found to give the best. Böcker has observed participation of the scaleni in only 2 cases, which were cut. While he did not meet with a failure by the Mickuliez method, the results from the Volkman operation, in spite of exact after-treatment and the wearing of a cervical collar in the severe cases, have not always been satisfactory. These poor results are due to the difficulties experienced in finding and removing the adhesions of the muscles to the surrounding parts and the deep-lying bands; and leads to the preference for the Mickuliez operation. The latter is simple in technique and exposes far better all deep-resisting structures, while it requires only a short after-treatment. Only a small wound is necessary and an extensive and deforming scar is avoided, so that the cosmetic effect is little worse than by the Volkman method. With the correction of the torticollis the cervical scoliosis which is frequently associated slowly disappears from the overcorrection. Only the worst cases will not be influenced. The Mickuliez operation is now employed in Hoffa's clinic, not only in the severe cases, but in the mild as well. It gives the surest and most complete success.

The Operative Treatment of Muscular Torticollis. An Answer to Dr. Doering of Gottingen.—Gerdes (*Zentralbl. f. Chir.*, 1907, xxxiv, 451, says that Doering's criticism of his (Gerdes) division of the scalenus in torticollis calls for a reply. That repeated occurrences have been observed after open tenotomy of the sternomastoid permits no serious doubt. The danger of recurrence and difficulty in the after treatment are the chief reasons for recommending the Mickulicz extirpation of the sternomastoid which is affected. Unfortunately this operation does not positively remove the danger of recurrence and has the disadvantages of a poor cosmetic effect. Lorenz considers tenotomy of the sternomastoid as a preliminary to the correction of a cervical scoliosis. How great the resistance to such a correction may be is shown by the fact that Lorenz took a half-hour to accomplish it in a rigid case, after a Volkman operation. In the majority of cases the resistance is due to the contracted muscles. Gerdes proposed to cut the anterior scalene, which is the most shortened of the muscles under consideration. This is a more rational procedure than a bloodless correction. After this tenotomy the correction is easily accomplished. Doering in his 37 cases did not bring the scalenus anticus muscle into the field in any case. The technique of tenotomy of this muscle is not difficult, as Doering maintains, but very simple, as everyone knows who has once performed the operation. Gerdes believes that when his operation is properly understood, because of its simplicity, it will be preferred for the moderate and mild cases. It renders particular after-treatment unnecessary and prevents recurrences more surely than simple tenotomy of the sternomastoid.

A Series of Cases of Goitre Removed under Cocaine Anesthesia.—Thomas (*Lancet*, 1907, i, 1143) says that ordinary goitres are fairly common throughout Wales and Monmouthshire, in England. In some counties they occur with great frequency, while in three counties they are rarely met with. In response to a circular letter 231 replies were received. One reply stated that in contrast to the common occurrence of goitres thirty years ago, they had, since the construction of the new water-works in his district, practically disappeared. Similar replies were received from other parts. A somewhat similar result from a changed water supply was shown in one family by removal from one district to another. The writer has operated on 55 cases under local anesthesia (cocaine 1 per cent.), including 4 cases of Graves' disease, and 1 case without anesthesia, without any mortality. Of 17 cases operated on under general anesthesia, including 5 cases of Graves' disease, 1 died from respiratory difficulties, 1 from hyperthyroidism (both Graves' disease), and 1 from sepsis. Thomas employs Kocher's collar incision. This was first outlined by a faint incision, along which one to two drams of a 1 per cent. solution of cocaine was injected by means of a Kocher syringe, from one end to the other of the incision, so as to form a continuous ridge. In order to cause as little deformity as possible, every divided muscle must be carefully reunited, the skin sewed by a very fine needle, and, lastly, the drainage tube and the skin sutures removed at the end of eighteen to twenty-four hours. Patients are allowed up on the third day and to go home on the sixth day.

THERAPEUTICS.

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The Treatment of Infantile Diarrhœa.—Perret (*La quinzaine thérap.*, 1907, vi, 450) advises careful feeding and intestinal irrigations with boiled water, 0.02 per cent. boric acid or naphthol solution, or normal saline solution. The irrigations facilitate the elimination of toxic substances from the intestine and supply the loss of the body fluids caused by the diarrhœa. During irrigation the child should be upon its right side; a soft, rubber catheter should be used and the receptacle of the fountain syringe, through which the lavage is given, should be elevated not more than five or six inches above the patient. The quantity employed should be from one pint to one quart and the fluid should be cold if the patient's temperature is elevated, hot if the latter is subnormal. As an intestinal antiseptic, three powders consisting each of 5 grains of bismuth salicylate and 2½ grains of benzonaphthol may be given daily. Lactic acid should not be prescribed if the patient is taking milk. If the child is less than one year old he may take a teaspoonful of the following mixture every half hour: lactic acid, 3 parts; quince syrup, 25 parts; distilled water, 100 parts. If older than one year dessertspoonful doses may be given. Bismuth in a mixture containing soluble bismuth phosphate, 2 parts; simple syrup, 10 parts; distilled water 90 parts may act well; the dose is one teaspoonful every hour. If vomiting is present, gastric lavage may be indicated. Injections of artificial serum are often very useful.

Prevention of the Formation of Gallstones.—Langheld (*The Therapist*, 1907, xvii, 4) states that the reformation of gallstones can be prevented in a very simple manner by exercising pressure upon the gall-bladder, through displacing the liver by about two inches, the activity of the viscus being thus considerably enhanced. The procedure is as follows: The patient, in loose clothing, sitting upright, leaning against the back of an arm-chair, places his arms on the arm-rests and takes a deep breath, holding it while counting 15, breathes out deeply and again counts 15 before inspiring again. Thus the liver is moved downward by about two inches. Undertaking the exercise for about fifteen minutes, morning, and evening is sufficient for the purpose. For those who have suffered from cholelithiasis it is wise to continue the measure permanently.

The Most Recent Treatments of Erysipelas.—HECHT (*Therap. Monats.*, 1907, xxi, 23) recommends the following methods of treatment: (1) Absolute alcohol is the most serviceable of the older agents, especially when a dressing which excludes the air can be applied, on the extremities for instance. (2) Salokrol and mesotan. These agents act well and may be employed if absolute alcohol is objected to by the patient. (3) Phenolcamphor has been warmly advocated. This consists of a mixture of liquid phenol, 30 parts; camphor, 60 parts; and spiritus vini 10 parts, and is a clear non-caustic fluid. Early in the infection it may be painted upon the affected part, later it may be applied upon gauze as a dressing. (4) A mixture of ichthyol, guaiacol, and oleum terebinthinæ 10 parts of each, spiritus vini 20 parts, acts as an antiphlogistic, an antipyretic and an analgesic, in erysipelas as well as in lymphangitis, furunculosis, etc., and also renders good service in acute rheumatic arthritis. Intravenous injections of collargol may also be employed.

The Treatment of Scar Formation Resulting from Gastric Ulcers.—EWALD (*Folia Therap.*, 1907, i, 1,) states that he has come to fear the cicatrices which follow gastric ulcers more than the causative lesion itself. The ordinary measures placed at our disposal—massage, electricity, baths, dietetic rules, etc., are worse than useless in these conditions; operative treatment has been our only resource, and by no means one certain to give permanent relief. It appears, however, that in thiosinamine we have a remedy which may possess a specific action on scar tissue. This agent is a derivative of the ethereal oil of mustard and has been repeatedly used in all postoperative troubles resulting from scar formation. Neisser has reported an instance of complete œsophageal stricture resulting from the ingestion of caustic soda which had resisted all forms of treatment for eight years in which a cure followed twenty-four injections of thiosinamine; no recurrence has occurred in nearly two years. Ewald has employed this substance in new formations of various kinds, especially benign stenoses of the œsophagus and somach and perigastric growths. In a number of patients no results were observed, but in others, subjectively, there was unmistakable improvement, while objectively there was decreased tenderness with milder symptoms, increase of weight, and ability to return to work. There was, of course, complete failure in malignant growths. An instance of gastric distortion following operation for cholecystitis and due to scar formation or pressure upon the organ by scar tissue and in which there was a palpable and tender tumor in the pyloric region was greatly benefited by injections of fibrolysine, a water-soluble combination of thiosinamine and sodium salicylate. The tenderness disappeared and other symptoms were relieved. The usual quantity injected contained 3 grains of thiosinamine to a dose. Thiosinamine itself may be injected in a mixture of 2 parts of this substance, 8 parts of glycerin, and 10 parts of distilled water.

Tincture of Strophanthus.—R. A. HATCHER (*Jour. Amer. Med. Assoc.*, 1907, xlviii, 1177) summarizes a paper, in which he discusses the advantages of strophanthus over digatalis, and cites results of experimentation with different samples of the tincture of the drug, thus: It may be stated positively that there is no difficulty in obtaining tincture of strophanthus in the open market of quite as uniform potency as obtains in the case of tinctures of other drugs which are not standardized, and that the seeds do not undergo deterioration on being kept several years, possessing in this respect a great advantage over digitalis leaves, which deteriorate rapidly. Digitalis continues to be of very variable strength, not only for the reason just stated, but owing to want of uniformity in time of collection, methods of drying and source, even when official requirements are met. It should be remembered that the strength of the infusion of digitalis varies greatly with the manner of preparation, an infusion made from the powdered leaf more nearly representing the full potency than one made from leaves which have been merely broken up in the mortar.

Differences in the Physiological Action of an Alkaloid.—O. H. BROWN (*Jour. Amer. Med. Assoc.*, 1907, xlviii, 1241) concludes a study upon this subject with the statements that the acid with which certain alkaloids, strychnine, quinine, and morphine are combined, alter decidedly the pharmacological action of the alkaloid—at least in regard to its toxicity for paramecia (the animalcules used for experimentation). The most toxic salts of strychnine and quinine are, in some instances, at least, those which stimulate catalytic processes most markedly. The salts of morphine with the smallest degree of toxicity, in some instances, at least, are the salts of those acids, the sodium salts of which stimulate catalysis the most markedly.

The Prevention of Arteriosclerosis.—HUCHARD (*Revue de thérap.*, 1907, lxxiv, 79) considers arterial hypertension an important precursor of arterial degeneration, and holds that the prevention of the latter condition consists, in great measure, in combating the former. In lessening the tendency to excessive vascular tension many means are at our disposal, the most important of which are: diet, muscular exercise, massage —particularly abdominal and precordial massage—baths—especially carbon dioxide baths—and the high frequency electric current. Of drugs, the most useful are the nitrites, among which are classed amyl nitrite, glyceryl nitrate, erythrol tetranitrate, sodium and potassium nitrite, with theobromine to assist elimination through the kidneys. Huchard considers renal insufficiency to be a very important early symptom of early arterial sclerosis and should command our best efforts for its relief, consequently, we should reduce to a minimum the alimentary toxins which are the chief cause of this difficulty. Here diet regulation plays an important part, as it also does in lessening the tendency to excessive arterial tension. The ideal diet is one composed chiefly of vegetables and milk and one from which sodium chloride has, so far as is possible, been eliminated. Diuretics also should be employed in order to increase elimination through the kidneys and it may become necessary to sustain the heart in the struggle against the peripheral obstacles. Heart tonics usually are unnecessary before the condition is well advanced but, when indicated, the intermittent administration of digitalin is advised. In fine, the treatment of what Huchard calls "presclerosis" consists in lowering arterial tension, combating toxic conditions, and increasing renal permeability.

Calcium Salts as Stimulants in Pneumonia and Cardiac Conditions.—Lauder Brunton (*Brit. Med. Jour.*, 1907, 1, 616) has prescribed these agents in pneumonia and in heart weakness of other types, and considers that the results obtained justify their further employment in threatened cardiac failure. In pneumonia he prefers to prescribe calcium chloride on account of its quick action. The dosage is from 6 to 10 grains dissolved in water, to which $\frac{1}{10}$ grain of saccharin to every 10 grains of the calcium salt has been added in order to disguise the taste. Such a dose may be given every four hours; it does not interfere with other medication which the patient may be taking. In cardiac conditions, in which a less rapid action is desired, he prefers calcium lacto- or glycerophosphate.

Creosote Carbonate in the Pneumonias of Children.—A. Fletcher (*Canada Lancet*, 1907, xl, 497) considers that the ordinary death rate from the pneumonias of childhood (25 per cent.) may be reduced to 5 per cent. by the administration of this drug, and that we owe it to ourselves and our patients to give the agent a continued and accurate trial. The method of the action of creosote carbonate is not thoroughly understood, but Fletcher thinks that it may be carried into the blood stream unchanged and is conveyed to the cells of the lungs to be excreted. The breath is soon laden with its odor and the skin also is concerned in its elimination. It this is true it is easy to understand the benefit the drug confers for it is brought into direct contact with the tissues in which the pneumococcus is developing. The dose for a newborn infant is 1 minim per hour; for an adult, 7 to 15 minims every two, three, or four hours. No gastric disturbance is likely to follow its exhibition, and it may be given dropped on lump sugar.

Intratracheal Treatment in Pulmonary Tuberculosis.—Mendel (*Revue thérap.*, 1907, lxxiv, 5) is very enthusiastic regarding the treatment of pulmonary tuberculosis by means of intratracheal injections of from 1 to 10 per cent. gomenol or eucalyptol in oil. Under this treatment, all the symptoms become ameliorated and the patient gains weight and strength. The method is applicable to patients afflicted with the chronic type of the affection as well as to those in the so-called pretuberculous stage. Of 200 patients in all stages of the disease, selected at random and treated by this method, Mendel reports 47 per cent. of apparent cures with return to active life; 33 per cent. of improvement, also with return to ordinary avocations, and 20 per cent. of failures; the last were individuals in the later stages of the disease. Usually the treatment must be continued for a considerable period of time.

The Comparative Antiseptic Power of Salicylic Acid and its Salts.—H. C. Wood, Jr. (*Univ. Penna. Med. Bull.*, 1907, xix, 312) concludes from a series of bacteriological experiments that the neutralization of salicylic acid, which itself is a very active germicide, by sodium or strontium greatly diminishes its bactericidal powers. This diminution is far in excess of the reduction in the amount of salicyl radical represented in the alkaline salts. It would seem that the antiseptics of the aromatic series are much more active in acid solutions. It has been shown also that the neutralization of benzoic acid gives the same results as that of salicylic acid. These results have a bearing on the use of these substances as food preservations, both sodium benzoate and sodium subsalicylate being used to some extent as antiseptics. The latter, Wood finds to possess distinct antiseptic properties, 1 per cent. solution being sufficient to prevent the development of microörganisms.

Cotarnine Phthalate.—C. A. von Ramdohr (*New York Med. Jour.*, 1907, lxxxv, 438) reports upon this drug as follows: Cotarnine is a derivative of narcotine, which is a constituent of opium, and its phthalic acid salt possesses special hemostatic properties; it is a yellowish, crystalline power, easily soluble in water, and its usual dosage is ¾ of a grain in tablet form three to five times daily. Even larger doses may be prescribed, and even then there are no unpleasant sequels. The particular field of its usefulness is in the control of uterine hemorrhage, especially that occurring in connection with fibromas. It may also be employed in dysmenorrhea and in the irregular menstruation of the menopause. It is less useful postpartum. Cotarnine is believed to act upon the vasomotor nerves of the uterus rather than upon the uterine muscle. von Ramdohr reports 6 instances of persistent uterine hemorrhage in which the ordinary remedies failed; 5 of these, including 2 of obstinate bleeding after abortion, responded quickly to cotarnine phthalate. He advises the use of this agent in selected patients who refuse operation, when a uterine hemostatic is demanded and there is no necessity for a hurried uterine contraction.

PEDIATRICS.

UNDER THE CHARGE OF

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Primary Intestinal Tuberculosis in a Nursing Baby.—Irving M. Snow (*Arch. of Pediat.*, April 1907, 297) reports an interesting case under this title. About March 1, 1905, a girl baby was born to healthy young parents, who had an older boy, aged one year, in good health. Early in April this boy died after an illness of six days, characterized by diarrhœa, high temperature, mucous and bloody stools, and terminal convulsions. As later events showed, this illness was probably tuberculous in character. The younger baby was much nursed by the mother and thrived for two months. At this time slight irregular fever began and continued until death, June 27. About June 1, a few papules appeared on the trunk and limbs, followed later by more papules and pustules on the thigh, forehead, and chest. A week later cough and occasional respiratory distress were noted, fever continued, and the spleen began to enlarge; bronchopneumonia developed and death occured after vomiting of blood and the passage of numerous bloody stools.

The autopsy revealed miliary tuberculosis of the lungs, spleen, and skin, caseous lymph nodes (bronchial and mesenteric), and a tuberculous ulcer of the ileum. Inoculation experiments with the mother's milk gave negative results. It was quite evident that both children were infected about the end of March, the older child dying quickly, the younger lingering for two months. Further investigation showed that the trained nurse, who had attended the mother in confinement, was at the time in poor health and coughed frequently. After a month's stay she went home and about July 1 tubercle bacilli were found in her sputum and lesions were discovered in her lungs. There can scarcely be a doubt that the nurse infected both babies by prolonged personal contact, either by kissing, sneezing, coughing, or breathing in their faces. It was also ascertained that a baby cared for by this same nurse nine months previously had recently died of bronchopneumonia.

In this case Snow believes that the avenue of infection and the primary lesion were in the digestive tract. The ileum, for about eight inches from the ileocecal valve, was firmly fixed in a mass of enlarged lymph nodes, and at this point was a circular area of ulceration of the mucosa, three quarters of an inch in width compressing the whole caliber of the intestine.

The Determination of the Caloric Value of Modified Milk.—GEORGE W. MOOREHOUSE (*Arch. of Pediat.*, February 1907, 86) gives a very simple method for estimating the caloric value of an infant's food, when the total quantity and the percentage formula are known. The method is as follows: Reduce the twenty-four hour amount to cubic centimeters, one ounce being equal to 29.5 c.c. Next determine the number of grams of fat, sugar, and proteids in the mixture by multiplying the number of cubic centimeters in the daily amount by the percentages of fat, sugar, and proteids. The calories from each constituent may be determined by remembering that a gram of fat furnishes 9.3 calories and a gram of sugar or proteid furnishes 4.1 calories. The calculation may be simplified by expressing the arithemetical processes by equations, thus: calories from fat $= Q \times 29.5 \times \frac{F}{100} \times 9.3 = Q \times F\ 2.74$; and calories from sugar and proteids $= Q \times 29.5 \times \frac{(S \times P)}{100} \times 4.1 = Q \times (S \times P) \times 1.21$. The sum of these two values gives the total calories furnished by the mixture, and this figure divided by the weight of the child in pounds gives the calories per pound per day.

In a companion paper by J. J. THOMAS (loc. cit., 82) a basis of comparison of the results obtained by Moorehouse's calculations is furnished. Quoting Finkelstein this author states that the average breast-fed infant draws daily during the first weeks of life one-fifth of its body weight; from the middle of the first to the end of the second quarter of the first year, one-sixth to one-seventh; and during the latter half of the first year, one-eighth of its body weight. Expressed in round numbers per kilo of body weight, during the first three months it draws 150 c.c.; during the second somewhat less; and during the third 120 to 130 c.c. Expressed in terms of energy quotient (Heubner), or calories per kilo, the requirement during the first three months is 100 calories per kilo (45.4 calories per pound); during the second three months, between 100 and 90 (40.9 calories per pound); during the latter half of the first year the requirement gradually sinks to 80 or a trifle below (36.4 calories per pound).

In regard to artificially-fed children, Heubner is of the opinion that the assimilation of cows' milk requires more work of the infant than breast milk, hence his energy quotient is 120. Czerny and Keller disagree with this and consider both milks equal in this respect. Finkelstein gives an energy quotient of 90 to 125 (41 to 56 calories per pound), but to be on the safe side and avoid any possible bad effects from over feeding, he prefers to use the same quotient for breast and cow's milk.

By using the calculations offered by Dr. Moorehouse it becomes a very simple matter to estimate the energy quotient of any milk modification, taken by an infant under observation, and to place the prescription of milk formulæ upon a scientific rather than a purely empirical, clinical basis.

The Vaccine Treatment of Tuberculosis in Children.—CLIVE RIVIERE (*Brit. Med. Jour.*, 1907, i, 859) has employed tuberculin injections in the treatment of tuberculosis of children, employing the opsonic power of the blood to determine the proper dosage and the proper interval between the injections. Before the injections the index was always lower than normal; throughout the injections it was kept at a higher point than normal. A high opsonic index is no contra-indication against the injections, particularly if the patient seems very low; the opsonic power is steadied, and though it may rise no higher, clinical improvement is usually made manifest. The adult dose is $\frac{1}{2000}$ mg.; children between ten and twelve may be given $\frac{1}{3000}$, at five years $\frac{1}{4000}$ mg., and at one year from $\frac{1}{8000}$ to $\frac{1}{12000}$; it is always safer to begin with a dose rather too small than too large. The intervals between injections are best kept at two weeks as long as the reaction is prompt. The index is examined before and after exercise, an injection is then given, and the examination made again in twenty-four hours, seventy-two hours, one, and two weeks afterward. The details of a number of cases are given, among them being cases of tuberculous dactylitis and superficial abscess, of tuberculous glands, of psoas abscess, of tuberculous joint disease, of abdominal and pulmonary tuberculosis. He sums up his article by stating that in tuberculin we have a most valuable remedy for localized tuberculosis. There is nothing dramatic in its effect, but with proper dosage a steady up-hill progress occurs both in the local conditions and in the general health. This improvement corresponds with an increase of opsonic power as shown by the alternate improvement and relapse in superficial lesions with the rise and fall of the opsonic index of the blood. Tuberculin is also of value in less localized infection especially if nutrition be not greatly impaired. With marked wasting and general illness no good results should be expected; yet, under such conditions, tuberculin may be the turning point in a down-hill course, so that unless the lesions are known to be widespread it is still worthy of trial. Tuberculin cannot take the place of surgical procedures, but should be used in conjunction with these. Caseous material must always be removed and this is best done when the opsonic index is high. If tuberculin is given sufficiently early, many cases would escape surgical interference. Secondary infection, always present with open tuberculous lesions generally lessens with improvement in the tuberculous process. Tuberculin treatment should only be undertaken by those who have given it special study.

OBSTETRICS.

UNDER THE CHARGE OF

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Retention of the Membranes through the Presence of Submucous Fibroid.—KREBS (*Arch. f. Gynäk.*, 1907, Band lxxx, Heft 2) reports the following case: The patient had a flat pelvis, and had been for some time in unsuccessful labor, the child perishing. Delivery was finally effected by craniotomy. A small piece of membrane was retained, and upon attempting to remove this it was found adherent to a small submucous fibroid. This was subsequently removed without especial difficulty, and with no hemorrhage. The patient made a good recovery.

MICHAUK'S case (*Inaug. dissert.*, Leipzig, 1866) was somewhat similar to the foregoing. His patient was a primipara, above the average age, and delivered by forceps. A piece of membrane was retained, and bleeding occurred. Upon investigation the uterus was found to contain a tumor the size of the fist, to which the membranes had been adherent. They were loosened and removed, and forty-four days after the patient's delivery the tumor was expelled in a necrotic condition through the vagina.

In BOISSARD'S case (*Bull. de la Soc. d'obst. de Paris*, 1902) a fibroid the size of a hen's egg was discovered after labor, to which the chorion was adherent near the placenta.

KNOOP (*Münch. med. Woch.*, 1903, No. 21), in a case terminating by spontaneous expulsion of the child, delivered the placenta and membranes by expression. In the membranes he found a fibroid tumor the size of a pigeon's egg. The mechanism of the adhesion of the membranes to these tumors seems to be atrophy of the decidua by the pressure of the tumor followed by adhesion of the membrane.

The Influence of Narcotics on Uterine Contractions.—KURDINOWSKI (*Arch. f. Gynäk.*, 1906, Band lxxx, Heft 2) investigated, by experiment and clinical observation, the influence of chloroform, chloral, morphine, scopolamine, and atropine, upon the contractive power of the uterine muscle. It was found that none of these had an appreciable effect upon uterine contraction. He also experimented with, and observed the effect of, Viburnum prunifolium. This drug has had considerable reputation as a remedy for checking uterine contractions. Kurdinowski found that in any but practically poisonous doses this drug, like the others mentioned, failed to effect uterine contractions.

The Formation of Decidua in the Ovary in Intra-uterine Pregnancy.—HORMANN (*Arch. f. Gynäk.*, 1906, Band lxxx, Heft 2) has demonstrated, and illustrates by microscopic photographs, the presence of fine intracellular fibers, syncytium and centrosomes, in the ovary during normal pregnancy. These observations may throw some light upon the possibility of ovarian pregnancy.

The Effect of Operation and also of Hemorrhage upon the Pregnant Patient.—Neu (*Arch. f. Gynäk.*, 1906, Band lxxx, Heft 2) reports the following cases: Case I, a primipara, between the fifth and sixth months of pregnancy, had repeated bleeding from varicose veins at the entrance to the vagina. Although her condition seemed otherwise good and there was no essential difficulty in the birth, which was spontaneous, the child was stillborn, and no apparent cause could be assigned for this except the repeated hemorrhage. In Case II a pregnant patient was found to have multiple cysts with nodules in the vagina. Excision was practised, followed by free bleeding, which required the use of a tampon to control it. This patient gave birth to a macerated fœtus. In Case III the vagina was almost occluded by the scars from a former labor. Operation was performed, and was complicated by hemorrhage. In this case the pregnancy remained without interruption. In Case IV, a multipara between five and six months pregnant had empyema of the gall-bladder, with peritonitis. Incision and drainage were practised and were followed by recovery. Spontaneous labor afterward occurred, after which the gall-bladder was removed and adhesions of the intestines liberated. This patient made a good recovery.

Innervation of the Uterus.—Roith (*Monats. f. Geburts. u. Gynäk.*, 1907, Band xxv, Heft 1) has studied the nervous supply of the uterus. He found upon minute dissection that the uterine muscle is stimulated by nerve fibers which arise from local ganglia in the connective tissue of the pelvis and also of the cervix.

Placenta Præviæ.—Burger-Graf (*Monats. f. Geburts. u. Gynäk.*, 1907, Band xxv, Heft 1) contributes an interesting paper upon this subject. In 44,676 births, 344 cases of placenta prævia were observed, being 77 per cent., or 1 in 130. The reports of other clinics give a frequency of from 1 in 58 to 1 in 1564. As regards age, placenta prævia is most frequent between the years of thirty-one and thirty-five. It is very much more common in multipara, 88.3 per cent. of these patients being multiparæ, and 11.7 per cent. primiparæ; 16 per cent. of the multiparæ were in the second pregnancy. Central placenta prævia is most frequent in multiparæ, and the tendency to this complication increases with repeated births. This variety is least frequent in primiparæ. In central placenta prævia only 31.3 per cent. go to full term; 16 per cent. were delivered before viability, and 52.6 per cent. had premature labor. In lateral placenta prævia the patient goes to term most frequently. Next to this comes partial placenta prævia, while central placenta præviæ naturally interrupts pregnancy most often. As to the time in gestation when central placenta prævia develops, it is most frequent in the eighth, ninth, and tenth months. Partial placenta prævia is most common in the ninth month, and lateral placenta prævia in the tenth month.

There seems to be some relation between placenta prævia and abnormality of the pelvis, as in 13.7 per cent. of these cases the pelvis was not normal. Twin pregnancy also predisposes to placenta prævia, and eight cases of twins occurred among the patients reported. As regards the presentation of the fœtus, the vertex presented in 69.2 per cent., transverse and oblique presentations occurred in 21.1 per cent., and breech presentation in 9.4 per cent. As might naturally be expected, the majority of these cases did not terminate spontaneously. In 81.3 per cent. some obstetric operation was necessary, while spontaneous birth occurred in the remainder. As regards treatment, Burger-Graf does not favor the use of the tampon, and in this he differs from many observers. The reasons lie in the fact that the tampon causes separation of the placenta, and thus increases bleeding. Its action is slow and uncertain, and the patient may lose blood and strength almost imperceptibly during its employment. In lateral and partial placenta prævia he urges the rupture of the membranes.

To prepare for delivery of the patient, he prefers the use of the elastic bag. In central placenta prævia, he would tear through the placenta, and then insert a bag distending it as completely as possible; 25 cases treated satisfactorily by this method are reported. He also employs combined version as being especially safe and efficient; 83.8 per cent. were delivered by this method, the average time consumed being two hours and nineteen minutes for each. In 99.1 per cent. of cases, bleeding stopped as soon as the version was complete. If the head was very low, and sufficient dilatation was present, he employed the forceps, but this occurred rarely.

As regards the management in the third stage of labor, in 70.5 per cent. the expectant plan was followd. In 9.4 per cent. the placenta was expressed by Credé's method; in 18.9 per cent. the placenta was manually delivered. After the placenta had been removed a gauze tampon was inserted in the uterus in but 7.3 per cent.

[We do not understand why the tamponing of the uterus with gauze was employed so rarely after the uterus was empty. In our experience, danger of hemorrhage occurs when delivery is complete. This bleeding comes from sinuses in the lower portion of the uterus and from vessels developed in the lower uterine segment with an abnormal attachment of the placenta. This bleeding can only be controlled by pressure, for the contraction of the upper expulsive segment of the uterus does not reach these cases. We believe it to be of great importance that the uterus, cervix, and vagina be thoroughly packed after delivery in all forms of placenta prævia, and in our experience, if this precaution be taken, postpartum bleeding does not occur.]

The general mortality among these cases was 5.85 per cent. So far as placenta prævia was concerned, it caused a mortality in central placenta prævia of 15.9 per cent.; in partial of 4.1 per cent; in lateral of 3.2 per cent; 8 cases of pyemia occurred, and 20 patients were delivered by operation, version being performed in 17. The puerperal period was afebrile in 88.8 per cent.; 5 of the cases had fever from causes not connected with the genital tract. The maternal morbidity is given as 9.6 per cent. Of the children, 45.1 per cent. were born living, and 54.9 per cent. were stillborn. Those children had the best chance of surviving who were born in spontaneous labor.

patient could not wear her rings for some months after her confinement. The sugar gradually disappeared from the urine without any modification in the patient's diet. The patient nursed her child, and the child did well. Marek considers this a case of increased action and secretion of the hypophysis, with increased adrenal secretion also. He remarks that cases of pregnancy having sugar in the urine may be divided into three groups: In one the sugar is milk sugar, which is very constantly found. In a second group the sugar is not milk sugar, but its presence in moderate quantity is not accompanied by a disturbance in the patient's health. Before conception occurred these patients had no sugar in the urine, and it disappeared soon after confinement. In a third group of cases, the patients have sugar in the urine before conception occurs, and it persists throughout the gravid state. The reviewer has recently had under his observation an interesting case of toxemia, accompanied by the prolonged presence of sugar in the urine. At the latter portion of the sixth month a multipara became drowsy, weak, and complained of excessive thirst. The urine showed a considerable percentage of sugar, with casts and albumin. There was some nausea and headache. There were also slight uterine contractions which threatened the interruption of pregnancy. These symptoms gradually subsided in severity under careful diet and constant and skilled nursing. In spite of a vigorous hygiene the urine did not become normal. The patient delivered herself at two hundred and ninety days of a living child, weighing 8.5 pounds, who shared in some degree the symptoms of the mother. The child's hands and feet were swollen soon after birth, its urinary secretion was deficient and highly colored, and its intestine contained much dark and disintegrated matter. Both mother and child improved steadily after birth.

The Mortality of Parturition.—UNTERBERGER (*Archiv f. Geburts. u. Gynäk.*, 1911, Band xcv, Heft 1) has endeavored to classify those conditions which most frequently cause mortality in parturient women. These statistics are gathered from a portion of the German Empire. He finds that 1 death in 488 births occurs from puerperal septic infection; 1 in 1480 births from tuberculosis; 1 in 1918 from eclampsia; 1 in 3454 from embolism; 1 in 3759 from postpartum hemorrhage; 1 in 4291 from placenta prævia; 1 in 8170 from nephritis in pregnancy; and 1 in 10,892 from atony and shock.

The Pelvic Kidney.—In the *American Journal of Obstetrics*, October, 1911, Bissell, of New York, contributes a second paper upon this interesting subject. From 1910, he reported the case of a patient whose pregnancy was complicated by dislocation of the kidney into the pelvis; and he adds other cases in his experience, aggregating 3 in all. These conditions may require Cesarean section at the time of labor, and can be successfully dealt with by a plastic operation, which in one or more steps replaces the kidney to nearly its normal position. The obstetrician must not lose sight of the fact that a pelvic tumor complicating labor, and obstructing delivery, may be a dislocated kidney, and that such a tumor should not be removed until no doubt exists concerning its nature. If it be the kidney, delivery should be effected by Cesarean section and the kidney replaced.

GYNECOLOGY

UNDER THE CHARGE OF

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Endometritis.—An attempt to gather together and unify the modern consensus of opinion on this much discussed subject is given in a recent article by Albrecht (*Monatsschr. f. Geb. u. Gyn.*, 1911, xxiv,, 397). The general principles of Hitchmann and Adler's work have been confirmed, he says, though the cyclic endometrial changes cannot be quite as sharply classified as those authors wish. The endometrium is subject to periodic changes due to the internal secretory activity of the ovaries, but the individual type of these periodic changes varies greatly. In principle it consists in an increase in the vital function of the corpus glands, evidenced by an increase in the volume of the epithelium, hypertrophy of the glands, and increased secretion, especially in their basal portion. The stroma takes part in the process by undergoing a decidua-like change. The development of these menstrual glands occurs irregularly in the uterus, and they do not all collapse and empty their secretion during menstruation, as Hitchmann and Adler claim, but some of them can persist throughout. This irregularity is due to the fact that the periodic functional activity of the ovaries, and also the responsiveness of the endometrium, are variable. It is, therefore, impossible to judge of the exact phase in the individual case from the appearance of the endometrium. Since "endometritis" means merely an inflammation of the uterine mucosa, and since the chief changes take place here in the stroma, the term "interstitial endometritis" is tautological, and should be given up. Etiologically endometritis may be divided into (1) bacterial, (2) toxic (due to chemical substances, necrotic tissue, toxins of infectious diseases, etc.), (3) mechanical and thermal. Since, however, all these causes produce identical conditions in the endometrium, a different classification is necessary for practical purposes. The most satisfactory from this point of view is that into (1) acute, (2) chronic, and (3) specific. In acute endometritis the alterative and exudative changes predominate over the proliferative. There is cloudy swelling, desquamatiom, even necrosis of the epithelium, swelling and increased secretion of the glands, as in all inflammations. Active hyperemia is present, with escape from the vessels of serum and leukocytes. The increased quantity of leukocytic infiltration in the tissue is the chief criterion of inflammation. Other infiltration cells (lymphocytes, plasma cells, etc.) are in the background as compared to the leukocytes in acute endometritis. This condition may heal or become chronic; that it so often does the latter is due to the unfavorable conditions for healing presented by the recurring increase in congestion and functional activity of the endometrium at each menstrual period. In chronic endometritis we have much less exudation of serum, or none at all; the cellular infiltrate siderable number of successful results following incision of the cervix and the subsequent use of alkaline douches.

Prochownick asserted that in one-third of all unfruitful marriages the husband has azoöspermia, resulting from a previous gonorrhœal epididymitis. One-third were due to gonorrhœal infection of the wife by the husband. In the remaining third pelvic inflammatory conditions play an even more important part than do neoplasms.

New Method of Ventro-suspension.—Liepmann (*Zentralbl. f. Gynäk.*, No. 6, 1907, p. 169) believes that Olshausen's method of suspending the uterus by the round ligaments is the ideal one. He modifies the latter's operation by drawing the loop of the ligament through a slit in the peritoneum and fixing it to the muscle by silk sutures. The advantages claimed for it are more permanent fixation, greater mobility of the uterus, and a more nearly physiological position.

Results of Myoma Operations.—Sarwey (*Arch. f. Gynäk.*, Band lxxix, Heft 2) reports 430 cases from the Tübingen Clinic in the course of seven years. The mortality after abdominal operations was between 4 and 5 per cent., after vaginal from 5 to 6 per cent., and after conservative operations about 6 per cent. Among 360 cases the tumor had undergone degeneration in 57 (6 sarcomatous), and in 104 cases there were visceral complications, cardiac degeneration being noted in 32. In 94 cases accompanying disease of the uterus and adnexa was observed. 300 patients were kept under observation subsequently. Nervous and atrophic conditions were seldom observed when both ovaries were removed (30.8 per cent.). Even when one or both ovaries was left typical climacteric disturbances were noted in 28 per cent.; hence Sarwey's inference that the benefit derived by the patient from sparing the ovaries after removal of the uterus is more than counterbalanced by the dangers of their subsequent degeneration.

In 25 cases of conservative myomectomy other tumors developed in 4; 3 patients became pregnant and 1 had rupture of the uterus at the site of the cicatrix. Of the 275 patients in whom a radical operation was performed none complained of the cessation of menstruation. Sarwey believes that the importance of preserving the functions of menstruation and conception has been exaggerated, and that the radical operation is preferable.

DERMATOLOGY.

UNDER THE CHARGE OF

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The Radiotherapeutics of Ringworm.—Sabouraud (*Brit. Jour. of Dermatology*, June, 1906) describes at some length the method of treating ring-worm by the x-rays employed at the St. Louis Hospital, Paris. The current used for exciting the Crook's tubes is derived from static machines and the tubes employed are self-regulating ones encased in strong steet-iron lanterns lined on the inside with a thick layer of ebonite. Metallic cylinders, all of the same length, but of varying diameters are used to localize the applications to the areas it is desired to depilate. The length of these localizers is so arranged that when the scalp is applied to the external orifice it is exactly 15 cm. from the anticathode. The areas to be rayed are carefully mapped off on the scalp with ink in such a manner that the same area is not rayed twice. Each patch is given a single exposure of sufficient length to produce fall of the hair without dermatitis. The length of the exposures is determined entirely by means of the radiometer invented by Sabouraud and Noire, pastiles of Bristol paper coated with an emulsion of platino-barium cyanide, which are placed midway between the surface to be rayed and the anticathode. From the beginning of treatment the entire scalp is daily painted with a weak tincture of iodine which insures local protection and prevents infection of healthy parts. If, through failure in the technique, a second exposure becomes necessary, this is only made after the expiration of a month. Since the adoption of radiotherapeutics the length of time required to cure ringworm has been greatly lessened. The average duration of treatment was formerly about twenty-seven months; it now may be as little as six weeks.

Origin and Structure of Moles.—Wilfred S. Fox (*Brit. Jour. of Dermatology*, March, 1906) states, in making concluding remarks on the subject: (1) That moles which show the typical column of nevus cells are epidermal in origin; (2) that there is a rarer variety of soft moles which show us typical nevus-cell arrangement, and whose origin is uncertain, possibly mesoblastic; (3) that the most cases of nevo-melanoma are nevo-carcinoma; (4) that melanomas do arise in the skin entirely apart from moles; (5) that Cohnheim's view of the origin of malignant growths is not borne out by the foregoing observations of the histology of nevo-melanoma; (6) that the pigment appears to be closely connected with the prime cause, by reason of which moles become malignant, whatever that cause may be.

Total Alopecia Following the Application of Cantharides.—P. F. Barton (*Lancet*, October 24, 1905) reports the case of a young man who presented a bald area the size of a shilling on the back of the head, which was considered to be of trichophytic nature rather than alopecia areata. It was painted with liquor vesicatorius (*Br. Pharm.*) twice in eight days. About eight days after the second application intense œdema of the scalp, face, neck, and upper chest set in. Soon there occurred a universal exanthem and hematuria. In the further course of the disease the hairs of the entire body and the nails fell out. At the end of nine months the nails grew in again, but the hair did not grow in, although the general health was restored.

The Treatment of Staphylococcic and Tuberculous Lesions by the Inoculation of the Corresponding Vaccines.—Thomas Houston and J. C. Rankin (*Proceedings Ulster Medical Society*, Belfast, December 5, 1906, reprint) give an extended article on this subject, including the method of preparing the test emulsions of staphylococcus or other similar microörganisms. The method adopted was that of Wright and Douglas, as described in their several publications. A series of cases of cutaneous diseases (acne, furunculosis, etc.) due to staphylococcus and to tubercle bacilli (lupus, etc.) are given, the results of treatment being in many instances strikingly favorable. The authors express themselves with caution and professional modesty, but the result of several years' experimental work incline them to agree with Wright's estimate of the value of this method, who states that "we have, in the power of raising the antibacterial power with respect to any invading microbe, out of all comparison, the most valuable asset in medicine."

The Blood in Relation to Skin Diseases.—Thomas Houston and J. C. Rankin, of Belfast (*Brit. Med. Jour.*, October 6, 1906), give the results of work done by them in which the blood, using Prof. A. E. Wright's methods of examination, gave important indications for treatment. One case, illustrative, may be quoted. The man had suffered for five years from staphylococcic lesions. It was found that his "opsonic" power was low, and that it could be raised by injections of staphylococcic vaccine, and that while his opsonic index was high the lesions always improved. He received seven or eight injections, which almost completely freed him from any cutaneous lesions, except a little persistent eczema on his wrists. Ten days' treatment with sodium lactate caused this to entirely disappear. After inoculations practically all the cases cited rapidly improved, though some relapsed; in a few the failure was obviously due to faulty procedure. The inoculation of the vaccine employed must be neither too large nor too small. Thus, in a case of acne with abscesses, inoculations with from 0.5 c.c. to 1.25 c.c. (used every day or every other day) of vaccine containing 1,200,000,000 dead staphylococci proved to be too large dosage, the curve of the opsonic index not being observed. In lupus if the opsonic index is persistently low the case will not do well with the Finsen light nor with any other treatment. If by carefully regulated inoculations, however, we raise the opsonic power, the improvement is marked. In those dermatoses associated with serous effusions, in which the coagulability of the blood is found diminished, the soluble salts of lime have been rapidly beneficial.

The Perborates in the Treatment of Diseases of the Skin.— Herxheimer and Ipsen (*Arch. f. Dermat. u. Syph.*, Band lxxxiii, Heft 1) report their experience with the use of the perborates of zinc, magnesium, and sodium in the treatment of various diseases of the skin. These are new preparations which contain considerable quantities of oxygen, which is slowly set free when in contact with the skin. The zinc perborate was employed in salves and pastes containing from 2 to 25 per cent. of the salt. A large number of cases was treated with these salves and pastes, chiefly eczema in various stages. The authors conclude that the chief indication for the use of the remedy is found in dry eczema with infiltration, it being contra-indicated in all acute forms of the disease, and in the weeping stage of the chronic forms. Trial was made of the magnesium perborate with the view of avoiding the irritation which sometimes followed the use of the zinc salt, but there was but little difference between the action of the two. The perborate of sodium was found too irritating in eczema, but was employed with good effect in acne as a kind of paste made by adding a handful of the salt to a half-liter of water and allowing it to stand a day before using. This salt was also found useful in the treatment of hyperidrosis.

The Action of Radium on Malignant Neoplasms.—W. Deane Butcher (*Archives of the Röntgen Ray* No. 76) states that in his opinion the fact has been well established that small superficial neoplasms, such as lupus and cutaneous carcinoma, may be destroyed by the radiations of radium. "A wart, a lupous nodule, a syphilitic infiltration, a rodent ulcer, or a patch of carcinoma will simply wilt and wither away under the influence of the Becquerel rays, like an uprooted weed under the action of sunlight." Radium, like the Röntgen radiations, has a selective influence on young, immature, and rapidly growing tissue. It is difficult to explain the mechanism or to frame any reasonsable hypothesis which will account for this withering action of the ethereal waves on the elements of abnormal tissue. The first action of radium is apparently to cause a spasm of the bloodvessels, which may eventuate in permanent blanching of the part as in the treatment of nevi. Later there may be congestion, skin reaction, and still later telangiectasis, just as from an overdose of Röntgen rays. In short, the problems are the same as those of radiotherapy. Butcher thinks that a small, superficial, circumscribed lesion may be as accurately, certainly, and speedily attacked by radium as by Röntgen rays. It is by no means the case that the most powerful and costly specimens of radium are required for every case of radium therapy. Pigmentation of the skin has been known to follow exceedingly minute doses of the radium radiation. Wichmann, of Hamburg, found that pathological tissues, such as carcinoma, lupus, and fibroma, absorb a greater proportion of the rays than does healthy tissue. Radium succeeds in some cases when the Röntgen rays have failed to benefit. Even in the last stages of carcinoma, when ulcerative changes are rapidly spreading, one occasionally notes a marvellous arrest of the ulcerative process after exposure to the Becquerel rays, even in cases in which the Röntgen rays have proved of no value. Reference in the article is made to the recorded cases of Lassar, Robert Abbe, and MacKenzie Davidson in various diseases, especially carcinoma. The whole subject is full of interest and of promise.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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On the Origin of Anthracosis of the Lungs.—Since the publication of the work of Calmette's pupils, Vanste nberghe and Grysez, upon the intestinal origin of pulmonary anthracosis, the conclusions which they came to have been ardently disputed and by some denied. BEITZKE (*Virch. Arch.*, 1906, clxxxvii, 183) among others has repeated the experiments of Vansteenberghe and Grysez and has arrived at diametrically opposite conclusions. Beitzke comes to the same conclusions as did Arnold in 1885, namely, that anthracosis of the lung is caused by aspiration of coal pigment. Beitzke finds that when india ink is injected into the peritoneal cavity of guinea-pigs and rabbits, the pigment is taken up partly by the lymphatics of the omentum, partly by those of the diaphragm, and is carried to the lymph nodes above the diaphragm and under the sternum. When the cells of these glands are saturated with pigment, the particles of india ink pass through the glands into the thoracic duct, whence they are carried to the blood stream. A general pigmentation of the organs of the body follows, in which, however, the lungs are not nearly so extensively pigmented as the spleen, the liver, and the bone-marrow. Anthracosis of the lungs could not be produced by merely feeding rabbits, guinea-pigs, and a dog with coal dust mixed in their food; and following the administration of india ink to dogs through an œsophageal tube there was no pigmentation of the lungs except in one dog who struggled violently during the operation and certainly aspirated pigment. Rabbits fed through gastric fistulæ did not show the slightest sign of pigment in the lungs. On the other hand, aspiration of soot, with or without ligation of the œsophagus, always gave rise to pulmonary anthracosis. In the last experiments the bronchus leading to one lung was closed, and afterward the animals were allowed to inhale soot. The lung to which there was free access of air showed deep an extensive pigmentation, while the other lung, to which the closed bronchus ran, was partially collapsed and totally free from pigment.

Blood Formation in Severe Anemia and in Leukemia.—MEYER and HEINEKE (*Deut. Arch. f. klin. Med.*, 1907, lxxxviii, 434) have devoted much time to the microscopic study of the organs in lymphatic, myelogenous, and mixed-cell leukemias, as well as in cases of pernicious anæmia which were atypical, inasmuch as myelocytes and undifferentiated lymphocytes were seen in the blood. They conclude that lymphatic and myelogenous leukemia are very different processes; for in one there is a general growth of the lymphoid tissue of the body, while in the other the lymphoid tissue remains passive, and is even compressed by the newly formed myeloid tissue. These myeloid growths arise from the cells of the organs, but not from the lymphoid cells. In the spleen the myeloid cells develop from indifferent pulp cells. In the liver the myeloid growth corresponds to the blood formation in the fœtus, the white cells arising in the perivascular spaces, the red cells in the blood-vessels. The myeloid transformation in the spleen, liver, and lymph nodes was also seen in cases of pernicious anemia, and in one case of aplastic anemia it was present to a remarkable extent. This is to be considered as a compensatory process to carry on the function of the bone-marrow, which is in some way seriously affected. It is, in its nature, a distinct reversion to the embryonic form of blood formation. This process of blood regeneration outside the bone-marrow may be seen in certain cases of severe anemia following acute infection, and may be brought about in animals by the production of anemia by means of various poisons.

The Influence of Veratrine on the Cardio-inhibitory Action of the Pneumogastric.—Busquet (*Jour. de phys. et de path. gén.*, 1907, ix, 50), experimenting with injections of veratrine in the frog, finds that this drug throws out of function very quickly the action of the vagus nerve upon the heart. Two minutes after the frogs received veratrine, stimulation of the vagus caused but very slight lengthening of the systoles, and three minutes after the dose faradization of the vagus had no influence whatsoever upon the heart's action. In such mammals as the dog, the rabbit, and the guinea-pig the same results were obtained, though the influence of the drug was more fleeting than in frogs. Unlike the action of atropine, the heart rate is not increased. The experiments did not show whether veratrine acts on the nerve itself or on the muscle of the heart.

The Differential Diagnosis between Pneumococci and Streptococci—Richard Levy (*Virch. Arch.*, 1907, cxxxvii, 327), after experimenting with blood agar as suggested by Schottmüller, the inulin-serum media of Hiss, and the litmus-nutrose-agar devised by E. Fränkel, comes to the conclusion that with none of these culture media can one differentiate accurately all streptococci from certain types of pneumococci. In devising a new and surer method he has put to practical use some observations made by Neufeld, who has described a specific bacteriolytic action of bile upon pneumococci. Levy employs, instead of bile, sodium taurocholate. The test is very simple, and consists in mixing a 5 per cent or 10 per cent. solution of sodium taurocholate in bouillon with an equal amount of a bouillon culture of the organism to be tested. As a control, equal quantities of sterile bouillon and the bouillon culture are mixed. The tubes are shaken vigorously. If the organism belongs to the pneumococcus or streptococcus mucosus groups the turbid bouillon cultures become clear; if the organism is a streptococcus the culture retains its original cloudiness. This method he has tried on 35 strains, and claims that it is the most satisfactory one for the differentiation of these bacteria. He concludes that Streptococcus mitior of Schottmüller is not to be definitely distinguished from Streptococcus longus, and that Streptococcus mucosus is a variety of pneumococcus and closely allied to it.

Nicolle and Adil-Bey (*Ann. de l'Inst. Past.*, 1907, t. xxi, 20) in another series of experiments have arrived at much the same results as Levy. They find that bile obtained from several different animals and that sodium choleate cause prompt solution of pneumococci when added to cultures of this organism. A large number of bacteria are partially refractory and the streptococci and staphylococci completely refractory to the bacteriolytic action of bile.

The Experimental Production of Atypical Epithelial Growth and the Origin of Malignant Tumors.—Fischer (*Münch. med. Woch.*, 1906, liii, 2041) states that the almost regular occurrence in certain cancers of subepithelial inflammatory infiltration of the connective tissue led him to try to produce artificially some chronic inflammation under the epithelium which would cause an increased activity of the epithelial cells. After using various substances without much success he was finally able, by injecting a suspension of scarlet-R in oil under the epithelium of rabbits' ears, to produce a very remarkable picture. Besides the usual cellular infiltration, hyperemia, and giant-cell formation, interesting changes occur in the epithelium. After a few days increased mitosis is visible in the germinal layer, even atypical mitosis, and the epithelial layer increases in thickness. Soon the germinal layer sends out processes into the depths which reach out and surround the oil droplets. The connective tissue is seen to be studded with columns and nests of cells, in appearance like the cancroid pearls. The picture is histologically indistinguishable from that of a flat-celled carcinoma. Areas of cells frequently pierce the cartilage of the ear, and in one instance a nest of epithelial cells was seen growing in the lymph space. Fischer thinks this overgrowth is not a result of the inflammatory condition of the connective tissue, for other oils do not have the same effect, but a distinct chemotactic action of the scarlet-R upon the epithelial cell. The oil slowly becomes absorbed and epithelial cysts are formed; the cells become horny and cholesteatomatous structures are formed, which finally break externally and the contents are discharged. As closely as the histological picture resembles carcinoma we have not a true malignant growth. There is no destructive overgrowth, and when the attracting substance has been removed growth stops and the cells degenerate. Up to the present a chemotactic action has been established for the leukocytes only. For such substances as exert a specific chemotactic action upon a special group of cells Fischer introduces the term attraxines.

From this important observation Fischer develops numerous theoretical considerations relating to the origin and growth of malignant tumors. He agrees with the Cohnheim-Ribbert view of the histogenesis of tumors from aberrant cells, presuming the development of a specific attraxine to start the cells into activity and then to stimulate them to overgrowth. He lays great emphasis too upon the important role attraxines may play in the normal embryological development of the organs; and, indeed, considers them the cause of the embryological or postembryological aberrant cells from which, according to Cohnheim and Ribbert, tumors spring. The presence, of a great variety of attraxines which such views assume finds its analogy in the endless number of specific bodies being discovered in the blood. Ehrlich, in quite a different way, has come to the conception of an unknown substance, the "specific x-substance," in the blood necessary for the growth of transplanted tumors. Fischer identifies these with his attraxines. It has been shown too that tumors are transplanted with greatest ease upon animals of the same strain, and, as peculiarities in metabolism are the features most often inherited, Fischer sees in this another prop for his hypothesis.

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ORIGINAL ARTICLES.

DISPENSARY IDEALS:

WITH A PLAN FOR DISPENSARY REFORM BASED UPON THE ADOPTION OF THE PRINCIPLE OF RESTRICTED NUMBERS.

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I wish to propose a plan for the regulation of dispensary work in New York city, based upon the principle of the limitation of attendance in proportion to facilities for adequate treatment. I believe that the essential features of this plan might be adopted with advantage in Philadelphia, in London, or in any large centre of population and of poverty in England or in the United States, where the prevailing conditions in dispensary work are similar to those in New York.

I shall endeavor to show: (1) That certain evils are bound up with dispensary work as it is now commonly conducted. (2) That in the opinion of the New York State Board of Charities the carefully devised dispensary law of that State has already accomplished almost all that can be expected of it in checking dispensary abuse; and that notwithstanding this, the evil of overcrowding and all of the objectionable practices associated with it, are not notably diminished. (3) That the cure, or at least the effective treatment, of disease, especially the successful treatment of many so-called functional disorders (even at the cost of following patients to their homes for the study of their needs), is the proper aim of dispensary work; and that the ideal of really effective dispensary work imposes upon dispensaries obligations not generally recognized. (4) That the existing district nursing organizations in New York City deal with an entirely different class of patients from those that ask for dispensary relief. (5) That while a few New York dispensaries are now doing district work, this work is designed chiefly for the relief in their homes of patients who are too sick to come to the dispensary, and, except in isolated instances, is innocent of any intention of checking the development of disease by the promotion of better habits of living. (6) That in New York city the most advanced dispensary work is the work of certain tuberculosis clinics which have undertaken to carry their treatment and teaching into the homes of patients, and which have thus indicated their acceptance of the ideal of effective dispensary work. (7) That many dispensary physicians recognize the need of a reformation in the methods of dispensary treatment, and advocate the extension of the work of the dispensary into the home, wherever it appears necessary to strike at the causes of disease by methods more drastic than the use of drugs.

I purpose, furthermore, to inquire whether it is feasible for a large city like New York, with its multitude of dispensary patients, to adopt the higher dispensary ideals; and to ask by what means, if any, dispensaries can be persuaded to accept these ideals.

I hold that in dispensary work, no less than in hospital work, there are certain standards of method and achievement, to fall below which is to fail. While hospitals, as evidence of their efficiency, present figures representing the large percentage of their patients who are discharged as "cured," a different statistical method is followed in the out-patient department; here the subject of boast is commonly mere growth in attendance, and no attempt whatever is made to judge the work of the department by any real standard of efficiency. But the essence of the matter, the very core of the problem, is not how many patients are treated, but how well they are treated, and we must endeavor to discover and to apply methods which make for efficiency, or be content to have our dispensaries stamped as failures.

DISPENSARY ABUSE. The literature of dispensary abuse is voluminous. It shows, in the main, that the advantage offered by freedom of access to dispensaries is seized by persons not dependent on charitable aid; that partly in consequence of this, and partly because of the efforts of each dispensary to build up a large clientele, dispensaries are overcrowded; that the quality of the work done in dispensaries is low and that it does not tend to improve. Among recent publications on the subject, distinguished either by originality in form or by fulness or novelty of content, I shall briefly allude to two.

In 1903 the Hospital Association of Philadelphia appointed a Committee on Dispensaries and Dispensary Practice, for the purpose of examining into the conditions of dispensary work in Philadelphia. (A similar committee, appointed in New York or London, would have found analogous conditions to deal with.) This committee, for the purpose of its inquiry, issued blanks asking for opinions upon existing evils, and calling for remedies. In response to these inquiries, replies were received from 187 physicians and from the officers of 39 out of 61 of the dispensaries then existing in Philadelphia.

From so many critics a great variety of opinion was naturally elicited; but putting aside, as having little bearing upon the purpose of this article, all peculiar and idiosyncratic views, and omitting also replies dealing merely with minor phases of out-patient practice, dispensary abuse was shown to group itself into three main divisions: (1) Abuse on the part of the patient: the use of dispensaries by patients able to pay private physicians. (2) Abuse on the part of the profession: careless and ineffectual treatment. (3) Abuse on the part of the dispensaries: encouragement of large and indiscriminate dispensary services (this in turn fostering the abuse of dispensaries by patients and physicians).

In the second paper to which I shall refer, Richard C. Cabot, proposing the improvement and extension of treatment in out-patient departments, thus sums up the abuses which limit the therapeutic usefulness of dispensaries: "The two great abuses are—first, hurry and consequent makeshift; and second, wholesale drugging," both of which, let me say in passing, are direct products of that encouragement by dispensaries of large and indiscriminate services, which began in London more than sixty years ago and which today is quite as common in this country as in the English capital.[1]

A descripiton of the quality of the work done in the out-patient departments of London, which applies literally to the work done in New York, was given by Mr. Timothy Holmes:[2] "As our out-patient departments are now conducted," says Mr. Holmes, "the patients get an average of the medical officer's time, which is reckoned by seconds rather than by minutes; they get a bottle of physic or some lotion or ointment, but there is no possibility of hygienic treatment, no provision for nourishment or stimulants. Now, much of the sickness, being due to intemperance, insufficient clothing, bad habits, bad food, and poverty, is not susceptible of this rough-and-ready treatment, but, so far as it is within the reach of treatment at all, must be treated at home. It is obvious that such medical service can not be satisfactory to the poor."

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[1] "At St. George's, Mr. Babington (Lord Macaulay's uncle) was, in 1829, appointed the first Assistant Surgeon to the hospital; those who had been so called previously having really been assistants to particular surgeons. Now began that lamentable competition which still continues between hospitals in the number of their patients. This number was easily accepted by the public as a test of the activity and usefulness of the hospital; and, as the number of in-patients could not vary very much for the same number of beds, competition could only be carried on by throwing open the doors of the out-patient department wider and disposing of the cases by hundreds an hour. Not only the general public, but the authorities of the Hospital Sunday Fund accept these numbers as evidence of merit and allow the share of the fund allotted to each hospital to be determined by this consideration." Thomas Mackay, "The State and Charity," page 180.

[2] Charity Organization Review, April, 1897

In an effort really to meet the needs of some of the patients applying for relief to the out-patient department of the Massachusetts General Hospital, Dr. Cabot has called to his aid a little band of social workers, and here, as well as in the tuberculosis clinics of New York, is found the promise of a brighter day for the dispensary patient of the future. "Believing that when a hospital or dispensary undertakes the care of a patient it ought really to care for him, and not be content with going through the form of doing it" says Dr. Cabot, "we have tried to fill the gap between good intentions and their fulfillment. To order for one patient a diet which he cannot possibly procure; for the next a vacation, which he is too poor to take; to forbid a third to worry, when the necessary cause of worry remains unchanged; to give to the fourth directions for an out-door life, which you are morally certain he will not carry out; to try to teach a fifth how to modify milk for her baby, when she understands, perhaps, half of what you say and forgets most of that half—this makes a morning's work not very satisfactory in the retrospect to anybody, and hardly more useful than the old-fashioned wholesale drugging."

It is to fill such needs as these, needs which typify the wants of a large proportion of the patients treated in the non-surgical departments of every general dispensary, and the needs of many of the patients admitted for surgical treatment as well—it is to fill such needs as these that Dr. Cabot has put into the field his force of volunteer social workers, who attend to such patients as the out-patient physicians of the Massachusetts General Hospital see fit to send them. The function of the social worker is to see that the directions of the dispensary physicians are carried out in the patient's home life, and to report back to the physicians any data concerning the domestic or economic life of the patient which bear on diagnosis or treatment. The results obtained at the Massachusetts General Hospital in the relatively small number of cases which there receive attention from the social workers stand out in bold relief against the common background of bad methods and worse results.

I shall not here enlarge upon the subject of dispensary abuse. The faults of our dispensaries are obvious, and the indictment against them is only too familiar. Nowhere else in all the wide field of voluntary or state aid for the unfortunate and the afflicted of our great cities is the gap between aim and achievement, between need and means, so wide as in overcrowded out-patient departments, where (in New York State) more than 2,900,000 consultations, examinations, or "treatments" are given every year, with a net social gain so small that the contemplation of it challenges the student's faith in the so-called philanthropy concerned in this work.

But it is idle to complain of the abuse of dispensaries by patients able to pay, unless effective machinery for the prompt detection of misrepresentation is established; it is useless to pretend to a desire really to meet the needs of the patient, so long as we accept as proper and sufficient the hasty examination and hurried advice which are commonly practised in our dispensaries today; it is futile to scold about carelessness on the part of physicians, so long as we ask the members of a volunteer and unpaid medical staff to deal unaided with overwhelming numbers in a few minutes or hours.

THE NEW YORK LAW. What efforts have already been made to introduce a better dispensary system and to check existing evils?

In 1899, in the State of New York, an attempt was made to stop dispensary abuse on the part of undeserving patients by the enactment of a State law requiring each dispensary to employ a registrar, to whom was assigned the duty of examining all applicants for dispensary treatment, for the purpose of determining their right to free medical relief. Under this law every applicant may be questioned in regard to his ability to pay for medical or surgical advice or treatment; if the registrar is in doubt, the applicant is required to sign a "representation card," stating his name, address, occupation, income, number in family, etc., which card is made the basis of a subsequent investigation. False representation by applicants for dispensary treatment is made a misdemeanor punishable by fine.

Shortly after the passage of this law it was stated by the Chairman of the Committee on Dispensaries of the New York State Board of Charities that the increase in dispensary attendance had been checked. The fact is that immediately after the enactment of the dispensary law, dispensary attendance actually increased, but at a rate less rapid than that of the growth of population. In 1906, however, the State Board was able to report an actual decrease in the number of persons treated in dispensaries in the State of New York, and especially in the dispensaries of Manhattan, although there was a slight increase in attendance in Brooklyn, the Bronx, Queens, and Richmond. The number of treatments given in dispensaries in the entire State during 1906 was 32,273 greater than in 1905, and presented a grand total of 2,927,351. In order to show the magnitude of the dispensary work done in the State of New York, let me quote here figures representing the number of persons treated, the number of separate treatments given, and the number of dispensaries in the State of New York in 1903, 1904, 1905, and 1906.

TABLE SHOWING THE WORK OF THE LICENSED DISPENSARIES IN NEW YORK STATE FOR THE YEARS 1903, 1904, 1905, AND 1906.

	Number of Dispensaries (1903).	1903.	1904.	1905.	1906.
NUMBER OF PERSONS TREATED.					
Manhattan	60	861,021	860,214	869,866	852,200
Brooklyn	31	115,248	110,480	111,259	117,445
Bronx, Queens, and Richmond . .	5	4,662	9,646	13,190	16,280
Total in New York City . . .	96	980,931	980,340	994,315	985,925
Outside of New York City	28	28,929	28,994	29,737	35,398
Grand total in the entire State .	124	1,009,860	1,009,334	1,024,052	1,021,323
NUMBER OF TREATMENTS.					
Manhattan	60	2,389,993	2,397,838	2,452,814	2,456,783
Brooklyn	31	282,394	289,678	300,448	311,809
Bronx, Queens, and Richmond . .	5	23,283	29,456	32,944	39,641
Total in New York City . . .	96	2,695,670	2,716,972	2,786,206	2,808,233
Outside of New York City	28	100,239	108,677	108,872	119,118
Grand total in the entire State .	124	2,795,909	2,825,649	2,895,078	2,927,351
NUMBER OF PRESCRIPTIONS.					
Manhattan	60	1,910,956	1,930,489	2,029,890	2,024,519
Brooklyn	31	200,247	188,017	195,573	198,419
Bronx, Queens, and Richmond . .	5	15,331	18,402	20,366	25,133
Total in New York City . . .	96	2,126,534	2,136,908	2,245,829	2,244,308
Outside of New York City	28	47,043	49,198	44,252	53,553
Grand total in the entire State .	124	2,173,577	2,186,106	2,290,081	2,297,861

Before passing judgment on the present New York dispensary law, we must inform ourselves on several points: First, what are the purpose and the main terms of the law; and, second, how far are the rules which it prescribes complied with. Both the requirements of the law and the extent of compliance with its various provisions (unimportant as well as important) are shown in the following table:

COMPLIANCE WITH DISPENSARY RULES OF 123 LICENSED DISPENSARIES IN OPERATION ON SEPTEMBER 30, 1906.

Rules.	Yes.	No.	In part.	Not applicable.
I. Public notice posted	120	3		
II. 1. Registrar	123			
2. Deputy (not required)	65	58		
3. Makes and preserves records	114	. .	9	
4. Receives applicants	123			
5. Sees that rules are enforced	23	. .	100	
III. 1. Examines all applicants	123			
superficially 25				
fairly well 84				
thoroughly 14				
are any refused admission	86	37		
a. Emergency cases admitted	120	. .	. .	3
b. Poor applicants admitted	123			
c. Doubtful cases admitted upon signing representation cards	91	32		
d. Subsequent investigation made	51	72		
e. Results of investigation filed	47	4		
f. Non-signers refused admission	52	1	. .	70
2. Representation cards in proper form	116	7		
3. a. Pass cards issued	122	1		
b. Penalty printed thereon	122	. .	. .	1
IV. 1. Matron	123			
2. Cleanliness and order preserved	121	. .	2	
3. Present at gynecological examinations	100	. .	. .	23*
V. 1. Contagious diseases excluded	123			
2. Registrar prevents exposure	123			
3. Registrar reports to health authorities	123			
VI. 1. Clinical or other instruction given	32	91		
2. Treatment conditional thereon	. .	32	. .	91
3. Consent of patient obtained	32	. .	. .	91
VII. 1. Apothecary (not required)	107	16		
2. Licensed or medical graduate	101	6	. .	16†
3. Appointed under Civil Service rules	6	. .	. .	117‡
VIII. 1. Board of Health ordinance observed	123			
2. Minute made before September 30	96	27		
IX. 1. Seats for all applicants provided	118	5		
2. Sexes separated in (a) waiting rooms	93	23	. .	7
(b) treatment rooms	107	9	. .	7
3. Suitable for equipment and supplies	118	1	4	

* Such examinations not held in these dispensaries.

† No prescriptions compounded in these dispensaries.

‡ Applies only to eight dispensaries connected with municipal hospitals in New York City.

The New York State Board of Charities, in a recent report, calls attention to the fact that compliance with what it regards as the "crucial point" in the proper administration of dispensaries, viewed from the social standpoint, namely, the examination by the registrar of applicants for treatment, is not uniformly thorough. In 1906, the examination by the registrar of applicants applying for treatment was reported as being done thoroughly in 14 dispensaries; in 84 dispensaries it was reported as being done fairly well, while in 25 dispensaries the work was found to be done only superficially. "It would seem," says the Board, "that the doors of the dispensaries of the State are reasonably well guarded in a large majority of cases; that they are partially protected in most of the remaining instances, and that they are not wholly unguarded, except in a very small number of cases." We are left to infer that the full and complete operation of the "crucial point" of the law, that is, the inquiry into the social status of applicants by the registrar, will still further, but in a relatively slight degree only, check the increase in dispensary attendance; but that any marked decrease in dispensary attendance, and hence, with the facilities at present existing, any better opportunity for close attention to the individual case, can result from even the most rigid application of the present law, is not to be hoped for.

It is beside the purpose of the present article to attempt to correct the published figures in regard to dispensary attendance, although it must be recognized that statistics of dispensary attendance cannot be accurate so long as dispensaries differ in their methods of recording patients, visits, and treatments, and so long as there is no method of determining the number of "rounders" registering simultaneously or successively in more than one dispensary. But whatever the percentage of error in the figures gathered by the State Board, the facts remain that the existing dispensaries of New York are, in the main, overcrowded; that under conditions of overcrowding and hurry, standards of treatment are inevitably low; and that after seven years of legal prohibition of the use of dispensaries by patients able to pay, but with *no plan or machinery for the routine registration and investigation of all cases* the general level of dispensary work is not materially higher than before; and the crying evils of overcrowding, superficial examination, and careless and ineffectual treatment, are unchecked.

The dispensary law of the State of New York is a conservative measure, drawn by men fully cognizant of the facts in regard to the abuse of dispensaries by a socially undeserving class; familiar, apparently, with the fraudulent practices of so-called dispensaries conducted for private gain under the cloak of charity; and informed as to the tendency of not a few dispensary managers to be careless about certain details of hygiene and to overlook certain considerations of decency in the grouping of the sexes and in the examination of female patients.

But the framers of the law, while seeking to suppress fraud and misrepresentation, were evidently desirous of interfering as little as possible with the managers of dispensaries honestly philanthropic in motive. So far as it is possible to judge from the terms of the law, there was no thought, in the minds of its framers, of compelling dispensaries to limit their work to dimensions consistent with their facilities; no idea that a system could be and should be worked out, by which applicants for dispensary relief would be compelled in every case to prove their right to the relief offered them. And yet it is plain that there is a sort of swindle, unconscious perhaps, but none the less real, in the attempt to make a relatively small dispensary organization meet the needs of an unlimited number of patients; and the lesson of the need for and the value of routine investigations of applicants for relief, long ago taught by the modern school of philanthropy, is today a recognized feature of all kinds of relief work except that of hospitals and dispensaries.[3]

By comparing with the present New York law a list of the remedies for dispensary evils suggested to the Committee of the Philadelphia Hospital Association, we shall see at a glance to how great an extent the New York law conforms to the existing state of current critical opinion.

Remedies Suggested by the Philadelphia Committee.	New York Law.
(*a*) Cards to be issued by the clerk under direction of dispensary.	(*a*) Pass cards issued bearing copy of section of law declaring false representation by a dispensary applicant to be a misdemeanor.
(*b*) Cards to be issued for investigation.	(*b*) Investigation of all doubtful cases required immediately *after* first treatment.
(*c*) Patients to present certificate from minister or physician.	(*c*) Such certificates not needed where doubtful cases are investigated.
(*d*) Refuse those able to pay.	(*d*) This is the purpose of the New York law.
(*e*) Fewer dispensaries.	(*e*) All dispensaries must be licensed by the State Board of Charities. Since the enactment of the law a number of applications for licenses have been refused.
(*f*) Cards to be issued by persons in charge of a district.	(*f*) All investigations are conducted by the Charity Organization Society, and Manhattan and the Bronx are treated as one district.
(*g*) Cards to be issued after patient signs statement of need.	(*g*) Provided by the law.
(*h*) Charge those able to pay for medicines.	(*h*) The law forbids any charge, whether for treatment or medicines, which shall have reference to the value of the thing furnished. Theoretically, persons able to pay either for treatment or medicines, are expected to obtain private aid. The charge of 10c. made in most New York dispensaries for medicales is a nominal charge, theoretically, having no relation to the value of the service rendered or the medicine furnished, though in most instances probably covering the actual cost of the medicine itself.
(*i*) Investigate homes of patients.	(*i*) Such investigations provided for in doubtful cases only.
(*j*) Signs in dispensaries announcing that treatment is for the sick poor only.	(*j*) Dispensaries required to post conspicuously in reception rooms, notices stating that the dispensary has been licensed to furnish relief to the sick poor who are unable to pay for same.

While the New York law has been of undoubted benefit, its most important public services have been the abolition of the "fake" dispensary through the use of the licensing power, and the gradual promotion of order, decency, and cleanliness in legitimately conducted institutions. The law has brought about no radical reform in the manner of conducting dispensaries; it has not raised, nor has it sought to raise, the standard of therapeutic achievement; nor has it attempted to bring about a more perfect correlation between dispensaries, hospitals, and the various relief agencies capable of playing an important and helpful role in improving the health of dispensary patients. It has not provided, nor has it attempted to provide, a complete system for the automatic exclusion of improper applicants; it has not eliminated the "rounder;" it has not made overcrowding impossible, nor has it insured the continuous development and improvement of dispensary work.

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[3] Since the beginning of the present year (1907) Mt. Sinai Hospital has employed an investigator to inquire into the validity of the claim for free treatment of every in-patient admitted to its public wards. Of the first 1500 free cases investigated, 45 were found to be able to pay seven dollars per week for an average of three weeks' treatment. Seventy others were classed as doubtful, but declined to pay.

THE DEMAND FOR EFFICIENCY IN DISPENSARY WORK, AND THE CONDITIONS UNDER WHICH EFFICIENCY MAY BE ATTAINED. It must be conceded that dispensary work will always be poorly done while the leading dispensaries, doing the bulk of the work in the city, are overcrowded as at present; that the elimination of undeserving applicants (not the occasional and accidental detection of misrepresentation, but actual knowledge and use of the knowledge of the financial status of all applicants) would aid in reducing the number of dispensary cases to practicable proportions; that such elimination cannot take place so long as patients are free to roam over the city from dispensary to dispensary without fear of detection or punishment; and, finally, that some method of limiting the number of patients in proportion to dispensary facilities, must be introduced as the basis of any really effective reform.

Let us not suppose, however, that we have merely to establish conditions under which careful diagnosis and intelligent consideration of the needs of each patient may be accomplished. The establishment of these conditions will at once bring into the foreground the importance of environment as a factor in the causation and cure of disease; then will follow a demand for the creation of machinery for the study of the patient's environment, and, finally, a realization of the necessity of extending the work of the dispensary, in suitable cases, into the home and the workshop. Appreciation of the influence of environment in disease has already led to the organization of social work on a large scale in the battle against tuberculosis; to the organization of societies for the promotion of tenement house reform; to the movement for the regulation and control of injurious trades. While it is the function of these latter organizations to deal with questions of public health from the standpoint of public prophylaxis, it is the place of the dispensary to seek out and to deal with conditions of living injurious to the individual patient.

Dr. Cabot has divided into two groups, which he terms, respectively, "organic" and "functional," cases commonly treated in the out-patient department of the Massachusetts General Hospital in 1904. In the group of functional diseases he has included constipation, dyspepsia, debility, neurasthenia, headache, and other pains without known cause, hyperchlorhydria, alcoholism, and other conditions in which the patient needs above all else, a correction of faulty habits and the practice of hygiene. These reforms are not easily or quickly won. "To obtain them there may be needed" (I quote Dr. Cabot's succinct recommendations) "sometimes careful instruction both at the dispensary and in the home; sometimes the patient must be removed from the friction and worry of home conditions and started afresh elsewhere (vacation, change of scene, convalescent home); sometimes he must be helped to get the necessary but somewhat expensive means of recovery—a set of false teeth, an abdominal supporter, a nourishing diet, a vacation." Here we have in a nutshell the problems which will have to be met in the future by dispensaries which aim at genuine amelioration of the condition of every worthy applicant for relief.

THE VISITING NURSE AND THE DISTRICT PHYSICIAN OF THE PRESENT DAY. The association of home visiting with dispensary work is not a novelty. District visiting by medical men on behalf of dispensaries, historically antedates by far the now popular independent district nursing movement. Of late a few of the dispensaries of New York, especially those engaged in tuberculosis work, have developed a system of home treatment or supervision in connection with dispensary cases. Before undertaking to describe this work, which in its principles and methods furnishes a model for dispensary work generally, let us glance for a moment at the work of the district nursing organization as it is conducted in New York today.

Miss Wald, of the Nurses' Settlement, states the need of district-nursing organizations in the following words: "District nursing, or a comprehensive system of care for the sick poor in their own homes, demands serious consideration, because the hospitals of the city are admittedly able to care for less than one-tenth of the sick of the city. On account of the high cost of hospital care, it is useless to hope to approximate the needed relief of the sick by hospitals, and other methods are thus brought before the community. There must always be great numbers for whom home care is impossible—patients who, regarded from the medical viewpoint, require the service that the hospital alone can give; but it is also true that their [hospital] places are frequently appropriated by patients who might be cared for in the home if proper nursing could be secured."

While the Nurses' Settlement, the leading and perhaps most highly-developed organization of its kind, coöperates with private physicians as well as with hospitals, dispensaries, and other charitable agencies of every description, an examination of the report of cases treated by the Nurses' Settlement shows that the admirable work done by this and other district organizations of similar scope, does not reach any large number of those dispensary patients who most need to be followed to their homes.

In 1905, 5032 cases nursed by the Henry Street or Nurses' Settlement, were classified as follows:

Unclassified medical	1735
Unclassified surgical	602
Pneumonia and bronchitis	956
Tuberculosis	296
Gynecological	212
Burns	197
Rheumatism	158
Obstetrical normal and abnormal	155
Meningitis	123
Typhoid fever	122
Contagious	119
Ulcers	118
Cardiac	97
Eye diseases	90
Alcoholism	6
No illness	46
Total	5032

Further light is thrown upon the character of these cases by the report of their final disposition, which I quote as follows:

Cured	2624
Hospital	740
Dispensary	598
Investigations	342
Died	312
Special nurse	165
Department of Health	69
Carried over in 1906	182
Total	5032

This report shows how close is the contact between the district nursing association and other relief organizations; it shows also that while nurses identified with district-nursing organizations are in touch with thousands of homes where medical advice and nursing aid are needed (and are, no doubt, given freely and wisely), these nurses are called more especially to the homes of acutely sick persons, and are only to a very limited extent in touch with the many thousands of dispensary patients suffering from disorders of the functional variety or from chronic organic disturbances associated with mild if depressing symptoms diseases directly traceable to or seriously influenced by habits of living. Hence, if the dispensaries are to undertake to develop their work along the most effective lines, they must build up district organizations of their own, must follow patients from the dispensaries to their homes, and there endeavor to inculcate habits of life consistent with health. Now, aside from the more recently developed work of the tuberculosis clinics, the home visiting, so long established in connection with the older dispensaries of the city, is much narrower in its purpose than the kind of visiting here proposed.

There are in New York city today fourteen dispensaries having outdoor or visiting physicians and nurses. Years ago, the lower part of Manhattan Island, then the city of New York, was subdivided into districts by six dispensaries, each of which appointed one or more district physicians. The duty of these physicians was to visit patients who were reported by their friends at the dispensary as being too sick to call there for treatment. The Good Samaritan (then the Eastern) Dispensary undertook to took after the district east of First Avenue and south of Fourteenth Street; the De Milt Dispensary assumed charge of the district on the East Side lying between Fourteenth and Fortieth Streets; the Northeastern Dispensary, the district included between Sixth Avenue and the East River, between Fortieth and Eighty-sixth Streets. The West Side was similarly subdivided, the southernmost portion being assigned to the New York Dispensary, the central portion to the Northwestern Dispensary, and the northern extremity of the city, at that time extending to Eighty-sixth Street, to the Northwestern Dispensary. The system then established has been continued to this day without change of district assignment, regardless of the fact that the Nurses' Settlement and other agencies, too numerous to mention, have since entered the field ready to share the burden. Indeed, but for the birth and growth of these new organizations, the dispensaries mentioned would have been overwhelmed by the increasing demands of a rapidly multiplying population.

The extent of the district work of the eleven New York dispensaries now registering the largest number of home visits (whether by physicians or nurses) is shown in the following table, compiled from the official reports:

	No. of patients visited in homes.	Total No of visits.
New York Infirmary for Women	1396	11,272
New York Homœopathic Medical College	1912	8,092
Northeastern Dispensary	2847	8,370
Northwestern Dispensary	5061	11,115
Northern Dispensary	2915	5,779
Presbyterian Hospital (including visiting for the Tuberculosis Class of the Vanderbilt Clinic)	5080	16,641
De Milt Dispensary	2283	8,193
Good Samaritan Dispensary	5007	6,554
New York Dispensary	1447	3,093
Vanderbilt Clinic	829	7,149
Eclectic College Free Dispensary	1216	3,167

I am indebted to eight of the dispensaries now doing district work for the information contained in the appended table:

Name of dispensary.	Date of beginning of district or home work.	Number of persons engaged in such work. 1. Physicians. 2. Nurses. 3. Others.	How many receive salaries? 1. Physicians. 2. Nurses. 3. Others.	Are salaries paid, if any, drawn from the general funds of the dispensary, or are they contributed by coöperating societies or individuals?
Flower Hospital Dispensary	1899.	11 physicians, 2 medical students.	None.	General Funds.
Northern Dispensary	1828. ?	4 physicians, 1 nurse.	4 physicians.	General Funds.
New York Dispensary	Before 1887.	2 physicians, 1 nurse.	2 physicians, 1 nurse.	General Funds.
New York Infirmary for Women and Children	1853 (by Dr. Elizabeth Blackwell).	1 nurse.	None (pupil nurse).	General Funds.
Northeastern Dispensary	1862.	2 physicians.	2 physicians.	General Funds.
Good Samaritan Dispensary	1892.	1 physician, 1 nurse.	1 physician, 2 nurses.	General Funds
Vanderbilt Clinic	February, 1903.	4 physicians, 5 nurses, 1 social worker.	5 nurses, 1 social worker.	Individual contribution.
Presbyterian Hospital Dispensary . . .	February, 1904.	20 physicians, 6 nurses, 2 social workers.	3 nurses.	From special fund

Name of Dispensary.	Is the home or district service intended exclusively for the benefit of patients who are too sick to call at the dispensary?	Are the physicians and nurses engaged in district work expected merely to give immediate relief to patients, or are attempts made to aid the patient to attain a higher plane of living?	Are funds for the relief of patients in distress at the immediate disposal of the District Staff?	Does the District Staff regularly coöperate with relief agencies other than hospitals?
Flower Hospital Dispensary	Exclusively for patients too sick to call.	Sole aim to heal the sick; social work not attempted.	No.	Not regularly.
Northern Dispensary	Exclusively for patients too sick to call.	Physician's chief duty medical relief; would also observe hygienic conditions.	No.	No.
New York Dispensary	Exclusively for patients too sick to call.	Physician's chief duty medical relief: would also observe hygienic conditions.	No.	With Charity Organization Society at times.
New York Infirmary for Women and Children	Exclusively for patients too sick to call.	Contact continued until patient is well; patients to come to dispensary when able.	Yes.	Use own funds usually; sometimes call in the "diet kitchen."
Northeastern Dispensary	Exclusively for patients too sick to call.	Sole aim to give physical relief —medical or surgical.	No.	Yes.
Good Samaritan Dispensary	Exclusively for patients too sick to call.	Reports Charity Organization Society.	No.	Yes.
Vanderbilt Clinic	Instruction of family of tuberculosis patients; and to dispensary patients unable to return.	Nurse in charge also represents New York Association for Improving the Condition of the Poor. Social worker advises as to employment, etc.	Immediate relief given by Asssociation for Improving the Condition of the Poor.	With United Hebrew Charities, Charity Organization Society, Association for Improving the Condition of the Poor, and fresh air agencies.
Presbyterian Hospital Dispensary . .	Volunteer physicians reach patients too sick to call and nurses reach other classes.	An attempt is made to improve living conditions.	Small fund available for immediate temporary relief.	Coöperates with large number of relief agencies.

The tabulation here given shows the scope of home visiting by medical men and nurses, as practised by dispensaries which have introduced these features into their daily work. The great value of the work done is unquestionable; its inadequacy fully to meet the needs of the dispensary population of New York city is equally beyond doubt.

THE NEWER AIMS OF THE TUBERCULOSIS CLINICS. Space forbids a full description of all the work now being done by the tuberculosis clinics of New York, including those of the Department of Health, Bellevue and allied hospitals. and certain voluntary institutions. In order to picture the aims of these clinics, I can do no better than to quote briefly the words of Dr. Linsly R. Williams, who directs the tuberculosis work at the Vanderbilt Clinic: "The work is educational, therapeutic, and humanitarian. The patient is taught by the doctor; and the patient and his family are taught by the nurse, who visits them in their homes, how to lead more hygienic and healthful lives, and, best of all, how to prevent others in the family of the tuberculous patient from taking the dread disease.

"In treating the poor, especially in a chronic relapsing disease such as tuberculosis, it is of the utmost importance for the physician to gain the patient's confidence and instruct him most carefully, to the minutest detail, how the patient may best care for himself in spite of bad housing and insufficient food.

"Treatment must be obtained for a patient in a sanatorium if he be an early case, in a hospital if he be far advanced and hopeless. In either case, the family is left penniless if the patient be husband and father, and means must be found to support them in the absence of the wage earner. This is a detail in which many of our New York dispensary physicians fail. A special effort is made at the clinic, by means of the nurses, to see that housing and food are obtained and maintained. The nurse is also of inestimable value in seeing that the doctor's orders are carried out, windows kept open, children sent out, sputum burned. Every means available to the New York tenement-house population is tried, to give the patient the most of fresh air and hygiene.

"The humanitarian value of the work is only a little removed from the therapeutic value, for is not treatment, or therapeutics, the attempt to relieve the sufferings of the sick, and to restore them to health in the quickest manner possible? Every therapeutic measure is really also humanitarian. If a patient, to keep up his strength and his ability to work, needs extra nourishment, milk and eggs are given him, for what good does it do to improve the appetite with tonics when there is no food to satisfy the hunger. When further relief is needed for rent, coal, supplies, etc., it is given by the New York Association for Improving the Condition of the Poor, the Charity Organization Society, or the United Hebrew Charities."

The work of the tuberculosis clinics illustrates principles and methods properly applicable to a large percentage of the cases which are now treated in the non-surgical departments of general dispensaries.

VIEWS OF DISPENSARY PHYSICIANS. I am able to present in outline the opinion of a number of physicians actively engaged in dispensary work in New York, who very kindly replied to the following letter of inquiry addressed to them on behalf of the Committee of Investigation of the Hospital Conference of the City of New York:

"DEAR DOCTOR: I am endeavoring to ascertain, for the Committee of Investigation of the Hospital Conference, whether or not it would be wise for the non-surgical departments of the dispensaries of New York generally to add to the work done, work to be carried on by salaried physicians and nurses in patients' homes.

"It is evident that in a large number of cases the ailment for which the patient seeks relief at the dispensary is directly or indirectly traceable to conditions of living or working which are more or less remediable; that the amelioration of these conditions can only be attempted when the conditions are known; and that they cannot be known unless the dispensaries can command the service of workers in the field.

"Incidentally, of course, the organization of dispensary work on the "district" plan would involve the registration and the home investigation of all applicants for treatment, and would thus enable the dispensaries to discover and to refuse treatment to patients who, under the prevailing conditions, abuse the privilege of free medical aid.

"May I beg for a reply to the following question: What proportion of the cases now treated in your department of the ——— Dispensary probably fall in the class of those who ought to be followed to their homes for the teaching and enforcement of hygienic living, and the prevention or cure of disease?"

The replies, in part, were as follows:

Dr. F. I. Wachenheim: "Perhaps a fourth or a sixth of my patients at the dispensary would be benefited by such visits as you mention; about a tenth should be treated in their homes, being not properly hospital cases, nor, on the other hand, proper subjects for ambulatory treatment. Another tenth are altogether unworthy of institutional aid and should be excluded by a better system of investigation; this last group has been steadily growing. The work should, of course, be paid for, but it is difficult to secure efficiency, at least at the salaries hitherto offered."

Dr. Sidney A. Stein: "Such a plan as you propose would be extremely useful. The class to which it would apply in our dispensary is very large: (1) The tuberculous cases; (2) the large number suffering from various chronic disorders for which there is no suitable hospital, even when suffering from acute exacerbation, such as cardiacs, nephritics, and diabetics; (3) I would estimate that at least 90 per cent. live under bad hygienic conditions, which could be at least ameliorated, if not done away with."

Dr. Hughes Dayton: "A very small proportion of the cases treated in the medical department of the New York Hospital Out-patient Department are those which ought to be followed to their homes as you suggest. Certain types of cases obviously require such attention, and we hope to accomplish this to some extent by securing the services of a visiting nurse. It is my idea that she should visit all tuberculous cases, cardiac and nephritic cases, whose condition is such that rest is essential, and who would be injured by the exertion of coming to the out-patient department more often than is absolutely necessary, and certain acute cases which demand confinement to bed and observation, yet for personal reasons, or on account of crowding of the wards, are not admitted to the hospital, etc. The idea of a salaried physician to follow such cases—preferably one of the out-patient staff who has already examined them—is one whose chief drawback is the difficulty of securing an appropriation.

"Concerning the disadvantages of the scheme of districting the city, it must be recognized that while systematic house treatment would allow investigation of applicants, and thus tend to decrease the abuse of free medical aid, it would, unless restricted closely, have two unfavorable effects. The ignorant class which fears and avoids hospitals, would be less than ever inclined to seek admission into the wards, if home treatment could be secured, and thus would increase the burden upon the district physician as well as decrease their own opportunities for recovery by remaining in unsuitable environments without constant nursing. Again, if the city were districted, and a certain class knew that salaried physicians could be obtained to care for them in their homes gratis, they would use and abuse the privilege rather than pay a private physician or make the effort to come to the out-patient department. If these objections could be overcome and the district physicians were authorized to refuse treatment at their discretion, much good might be accomplished.

"You speak of the necessity of house investigation of all applicants for treatment. How this could be done without great expense is difficult to see, considering the number of new cases applying daily. Of course this is the only way to stop the abuse of the dispensary."

Dr. Isador Abrahamson: "I endorse fully your recommendations, but would suggest widening the scope of patients' treatment. Patients suffering from the various so-called functional nervous diseases, including neurasthenia, hysteria, choreic habits, spasms, also the milder types of psychoses, lasting either a brief time or remaining mild for an indefinite period (phobias, compulsory ideas, hysterical psychoses, etc.); also various organic diseases of the nervous system where it is impossible for the poor patient to come to the dispensary or where essential portions of the treatment must be done at home; all these form even more gratifying objects as far as results are concerned than others that are taken care of in sanitariums, homes, and hospitals.

"Hospitals as a rule refuse to accept "nervous cases," partly due to their inability to give them the proper treatment. They are thus in a miserable plight—misunderstood at home, not accepted anywhere, they come to dispensaries where the lack of time prevents a thorough inquiry into those home conditions usually at the basis of their troubles. They require proper home instruction in cleanliness, proper feeding, the need of light and air—in short, hygienic rules; secondly, home instruction in various hydriatic methods; thirdly, home instruction in massage, in frictions; fourthly, instruction in care of the bed-ridden invalid, avoidance of bed-sores, etc.; and lastly, the explanation at length to the rest of the family of the nature of the illness, the need of forbearance, etc., especially in neurasthenia, hysteria, mild pyschoses, etc., and so on."

Dr. H. Schwarz: "Your proposal certainly represents one step in advance in the proper care of our ambulatory sick, but I would take the liberty of adding that it is not the first step that ought to be made. You mention "organized and paid work outside the dispensary;" this should first be brought about within the dispensary. There should be one or two heads to each department, who should be required to attend regularly *every day* and should be paid for so doing. The patients should have the privilege of coming every day if necessary and of seeing the same physician. On Sundays and holidays one man should be delegated to see the urgent cases, or those under daily observation. With this system of daily supervision our patients would not go from one dispensary to another, or from one to another department of the same dispensary, and they could be treated with greater success; in addition, the physicians attending the clinic would get better training in their respective branches. "Even with the above scheme in operation there is need for a service to investigate the mode of living, etc., of our patients. At least 10 per cent. of our patients would be better off for advice and practical knowledge in hygiene; home instruction is especially needed for the parents of artificially-fed infants."

Dr. M. Katzenberg: "The great majority of patients ought to be followed to their homes for the teaching and enforcement of hygienic living; but the only ones who should be helped and given good advice at their homes are the really worthy sick ones. There is need of a very thorough investigation of this dispensary question. Thorough work can only be done when the city is divided up into small districts and a reliable medical census taken."

Dr. George Mannheimer: "Fully one-half the number of our medical patients at the Mt. Sinai Dispensary ought to have home supervision. In the case of tuberculous patients the value of this measure is well understood. The tuberculosis dispensaries could not do their work without it. The majority of our patients suffer from gastro-intestinal disturbances, induced chiefly by faulty living. To correct this would be the proper treatment. But here is our difficulty. We do not know what our patients eat; how they prepare their food; what articles of food are accessible to them; under what names the ordinary foods that we recommend are known to them. Of course, we ought to know something about the sanitary condition of their homes and workshops, and about the nature of their occupations. Visiting physicians and nurses would greatly assist us by supplying this information and ameliorating these conditions."

Dr. James Morley Hitzrot: "We attempt in the New York Hospital, largely through personal efforts, to see that our cases of surgical tuberculosis have fresh air, etc., and are often able to correct their ideas of ventilation and diet with some benefit to them."

THE PROGRAM. In conclusion, I present the following program, which, if carried forward with a determination to stop at nothing short of the highest attainable efficiency, will create a new order of things in dispensary work, and through a well-organized and properly controlled dispensary system, will influence in a marked degree the health of the community.

(*a*) *The Principle of Restricted Numbers.* The first aim in any scheme of dispensary reform must be the prevention of overcrowding. Not until the number of dispensary patients to be examined is reduced to reasonable proportions can careful physical examination, proper history-taking, and accurate diagnosis, be expected.

The facilities of the existing dispensaries can easily be calculated; normal standards of attendance can be fixed, and the dispensaries can then decline to admit additional patients. If for any reason individual dispensaries continue to prefer quantity to quality in their work, and decline to impose voluntary restrictions upon themselves for the sake of the greater efficiency which would result from such restrictions, it will then be in order to request the State Board of Charities to replace its present unlimited license by a new form of license, stating the number of patients which a given dispensary, with specified facilities, will be permitted to admit for treatment.

If, when this has been done, it is found that more dispensaries are needed, private charitable enterprise will probably supply the deficiency; in the absence of such volunteer relief, the municipality must provide such dispensary facilities as are needed. It is doubtful, however, that additional dispensaries will be needed if the principle of restricted numbers is adopted as *one step only* in the scheme of reform; if, while seeking to establish new and higher standards in dispensary work, we at the same time encourage dispensaries to make better use of the buildings and the organizations under their control, and if we furthermore adopt an effective plan for the total exclusion from dispensaries of patients who owe support to the medical profession, and have no proper claim upon charity.

(*b*) *The classification of dispensary cases into* (1) *those needing dispensary relief only, and* (2) *those needing social aid or guidance.* With the acceptance of the principle of limited numbers (a principle as clearly necessary here as in the management of hospitals, the capacity of which is assumed to be fixed by the number of beds and the general resources of the organization), overcrowding and the indifferent work resulting from overcrowding, will cease; not until then can cases be separated into those two great groups whose different needs demand a splitting of the dispensary staff into two forces—one for duty in the dispensary, the other for work in the outer field. And what are these two great groups? The first, found chiefly in the surgical departments, consists of patients requiring dispensary relief only; the second, centred in the non-surgical departments, includes all patients who must be followed to their homes in order to effect a cure. Each dispensary, having classified its patients in the manner indicated, can then undertake to carry out that higher therapeutics, which does not content itself with iron and the unenforced recommendation of fesh air for anemia and debility, or with tonics or laxatives for obscure disorders generally, but which searches diligently for the causes of disease, and calls to its aid all the forces of the community capable of changing an injurious environment into a healthful one.

(*c*) *The Tightening of District Lines, and Its Influence in Controlling Misrepresentation.* It is safe to assume that, in spite of the inconveniences of urban travel, patients moved by habit, by sectarian bias, by the greater reputation of some dispensaries, or by dissatisfaction with the results of treatment, will continue to come to dispensaries from distant parts of the city. But just as dispensaries have imposed upon themselves voluntary limitations with regard to their present district or home work, can they gradually limit the residential district from which any patients will be accepted. By a voluntary understanding, dispensaries can thus lay the foundation of a system of district apportionment, ultimately to be established and regulated by law.

With district lines drawn tighter and tighter, misrepresentation by patients able to pay for medical treatment will become increasingly difficult, and a large number of those who now seek the aid of dispensaries, but who are able to pay for the services of private physicians, will find that they cannot escape detection. Misrepresentation will be automatically exposed if attempted by any of the patients falling within the group of district or home-visited cases; and the wrongful use of dispensaries will thus tend to diminish in proportion to the development of district work. The investigation of cases of doubtful financial standing, not requiring home treatment, may meanwhile proceed as at present; but the number of cases to be investigated will be materially lessened, and the percentage of those who can be reached by the machinery at present available, will be proportionately greater.

What the Final Organization will Include. Ultimately the city will be ready for the complete reorganization of its dispensary work under the guidance of the State Board of Charities, or a similar controlling body, upon a plan providing for the following:

1. Limited dispensary licenses, in the place of unlimited licenses, that is, limitation of the work of each dispensary to a suitable number of patients, to be residents of an assigned district.

2. Registration of all families claiming dispensary relief, and the refusal of relief (except in emergency cases) to all families not regularly registered and known, thus applying to dispensary work the methods, and perhaps, also, the machinery, of the Charity Organization Society and of other modern philanthropic associations, and while extending the scope and aims of the dispensary, at the same time restricting the aid given to those absolutely in need of it.

3. A system of home visiting, aiming at the effective treatment of functional and other disorders depending upon environment.

4. The use of the power of the State to prevent the embarrassment of existing dispensaries. This implies the encouragement of new dispensaries, voluntary or municipal, whenever warranted by pressure upon existing dispensaries.

5. Periodical readjustment of district lines to allow for shifting of population and for the enlargement of the facilities of established dispensaries. (On account of the varied character of established dispensaries, assigned dispensary districts at the outset will necessarily overlap; thus special dispensaries like those now devoted to orthopedic or to eye and ear work, with relatively extensive facilities of a certain kind, would be permitted to accept patients from districts greater than those assigned to large general dispensaries in the same locality having only limited facilities for the class of work carried on in the more highly specialized institutions.)

The reforms here proposed are restrictive and will inevitably meet with opposition; they will, no doubt, be considered especially objectionable by dispensaries which have a weakness for formidable statistics, and by those which, on account of certain college affiliations, are tempted to seek above all else a large attendance of varied cases—the larger the better for the purposes of medical teaching, no matter what the consequences to the majority of the patients, who make up the despised residue of every teaching dispensary of unrestricted growth. (I do not wish to be understood as opposing the use of dispensaries for medical teaching. The needs of some dispensary patients are at variance with the interests of schools controlling dispensaries, but both the medical student and the patient would be in a fairer way to get what they need, if each school were permitted to utilize several dispensaries instead of a single one.) But if we accept the principles upon which the proposed reforms are based, we cannot escape the duty of endeavoring to put them into effect. The theory of limited numbers in dispensary work is vital. I venture to say that there will be no real, no far-reaching, no general dispensary reform in our large cities, until this is conceded, and that, at last, the acknowledgment of the relation between facilities, on the one hand, and effective work, on the other, will prove the means of raising our dispensaries to the top notch of usefulness.

BENIGN STENOSIS OF THE PYLORUS.

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The following study of the symptomatology and treatment of benign stenosis of the pylorus is based on the records of 11 cases which came to operation in the New York Hospital. These cases, therefore, can be definitely separated from the more frequently occurring classes of pyloric stenosis, those due to malignant disease and to the scars of ulcer.

Stricture of the pylorus is difficult to demonstrate in the post-mortem specimen, unless the occlusion be developed to a very marked degree. There are no standard measurements of the normal pylorus in adults, similar to those which have been compiled for the gastric outlet in infants. The lumen of the pylorus varies so considerably in stomachs which are evidently healthy that it is almost impossible to formulate hard and fast rules to govern comparative tests, and the common method of testing with the end of the finger is perhaps as good as any other.

It is rather suggestive to find in comparing the case histories with the pathological studies of those cases in which hypertrophy and stenosis of the pylorus have been found at autopsy, that the condition had seldom been recognized clinically. It is a fair inference and usually true that these individuals have suffered with their last illness and have died of some disease other than the gastric condition from which they must have been afflicted for years. This fact suggests, and in a degree confirms, the statement that these stenoses are benign, not only that they are not growths of malignant nature, but also that a long life is not incompatible with their presence. This explains also why careful microscopic studies of the tissues from cases clinically diagnosticated are so few in number.

In the accounts of benign stenoses scattered through the literature numerous causal factors have been noted. Cruveilhier found a mucous polyp closing the pyloric orifice in one of his cases, and those tumors which Rokitansky examined and considered due to chronic gastritis were probably polyps of a like nature. Myxomas, myomas, and fibromas have all been described as causes of benign stenosis. The morbid changes in the structures of the pyloric ring which result in hypertrophy and stenosis usually assume one of two characters. There is either a marked increase of the glandular substance or a high degree of hypertrophy of the muscle, and one or both of these changes are found fairly constantly in specimens submitted to examination. The increased glandular tissue noted in these cases has the general architecture and appearance of Brunner's glands and is, as a rule, provided with canals leading to the mucosa.

These masses of gland substance make up at times considerable tumors which project into the pyloric canal. There is some reason for believing that this glandular growth is the primary and fundamental change which distinguishes pyloric hypertrophy; for when the muscular tissue is found developed to a marked degree, it is often associated with nests of these glands which have undergone an atrophy presumably from compression. The congenital pyloric stenosis of infants appears to be not of this nature, but to consist rather of a primary increase of the muscular layer and usually of the circular fibers. Thus Gran found in infants, in comparing sections of normal pylori with those in which there was marked hypertrophy, that the circular muscular layer was twice as thick as normal, while the longitudinal layer showed no increase. It is, however, notable that all the other tissue elements, even the serosa, were approximately double their normal thickness.

The somewhat varied morbid anatomy of these conditions has resulted in various theories for explanation, but none of them is conformable with all the facts. Von Recklinghausen's idea of the growth of uterine tumors has been applied to pyloric tumors by Meinel and Magnus-Alsleben, and while seeming to explain some cases, it is quite inadequate in others. Although it is not impossible that some of these cases of benign stenosis originate from a congenital abnormality which does not become manifest until adult life, the history elicited from such patients not only does not suggest any such condition, but rather, often indicates a primary functional derangement which by persistence could here, as in other situations in the body, finally produce a morbid anatomical state.

The physiological investigations of Cannon, Pawlow, and Starling have made it evident that at the pyloric valve there is one of the most delicate chemical and secretory adjustments of the body. The pyloric valve remains closed until the chyme in the pyloric end of the stomach is thoroughly disintegrated and acidified; then only does it normally allow free access to the duodneum, where now the acidified chyme causes a stimulation of constriction to the pylorus which persists until the alkaline bile and pancreatic juice have neutralized the acid. The reaction of the chyme is the adequate stimulus for the reflexes which contract or inhibit the sphincter pylori, and this same stimulus produces exactly opposite results in the sphincter according to whether it is applied to the gastric or to the duodenal mucous surfaces. This adjustment alone, even without that necessary absorption of "secretions" which stimulate the pancreatic cells, is so complex and nicely regulated that it can be easily understood how neurotic states and even mere errors in diet may overthrow the balance. The hyperacidity and anacidity that are commonly noted as temporary conditions in otherwise healthy individuals must demand considerable efforts at compensation on the part of the pyloric mechanism to produce the comparatively mild symptoms that characterize them.

There are so many factors which enter into the normal physiology of the pyloric function that we can but seldom find the break in the chain which marks the first deviation from the normal in any individual case. When we study cases of benign stenosis of the pylorus, however, those signs which clinically characterize pyloric spasm, that is, pain, increased peristalsis, and vomiting, enter into the histories of the early stages of the disease, and irregular attacks of this nature are often the only sign of disordered function which may occur during long periods of time, sometimes for years. As time goes on, the periods of pyloric spasm become more and more frequent. The condition, perhaps, is not unlike that of broken compensation in cardiac disease, in which each failure predisposes to the next, and each effort at compensation falls short of the condition attained to by the one before. The following history illustrates this clinical fact:

Case I.—Lawrence F., aged forty-six years. The family history is good. The patient says he has always been well and strong up to the present sickness, which began three years ago with attacks of vomiting lasting a week. During these attacks the patient vomited three or four times a day, the vomitus being food material and greenish fluid, never any blood. After a week in this condition the patient says his normal health would return and he noted no gastric symptoms in the intervening periods. Until a year and a half ago he thinks these disturbances occurred not oftener than every four or five months, but for the last year there has been an increase in their frequency and severity. Six months ago he grew so weak that he gave up work. He is now vomiting three or four times daily, has lost much in flesh, his appetite is poor, and his bowels very constipated. He has never vomited any blood, nor noted tarry stools. Examination showed a dilated stomach. The other organs appeared normal. At operation there was found a hard, thickened pylorus, the lumen of which admitted a 20° F. sound. A section of the pylorus removed at operation showed a marked increase of fibrous tissue.

This case presents a typical history of this disease. The recurring attacks alternating with shorter and shorter periods of normal health are often accompánied by a gradual change in the gastric juices as evidenced by the secretion of HCl, by signs of stasis and fermentation, and in some cases by absence of pepsin (Case XI). The periodicity of the process, slowly merging into a permanent disease entity, suggests the vicious circle, or functional habit, which characterizes so many pathological changes in their incipience and which continues until a morbid growth results. Even after correcting, as we believe, the anatomical defects in certain of these conditions by proper surgical means, health may not be restored, the vicious habit having become so established that normal gastric secretion is almost impossible.

The following patient was operated on twice and while improved was never permanently cured:

Case II.—Michael M., aged forty years, entered New York Hospital, November 26, 1890, complaining of periodical vomiting and constant gastric distress. His family and past history were unimportant. He began to have vomiting attacks five years ago, and there has been a slow increase in their frequency, until during the last two years he has been an invalid. On account of the constant distress and frequent vomiting he has lost flesh and strength. He has seen several physicians and the diagnosis of carcinoma has been made. Examination showed a dilated stomach, but no tumor. The gastric contents after test meals contained large amounts of free HCl; there were signs of stasis, but no blood. The stomach capacity was ten pints. A gastro-enterostomy was done; the pylorus was found enlarged and indurated; there were no signs of former ulcer. Again two years later a gastroplication was performed on the dilated stomach. At the last analysis made of this patient's stomach contents he still secreted excessive amounts of HCl, and there were evidences of fermentation.[1]

While it is true that many cases of this disease after proper treatment make a recovery to complete health with almost magical promptness, yet the class of which the above case is an example, is not extraordinary. In these the underlying neurasthenic habitus, the predisposition to abnormal functional activity, prevents a quick restoration to the normal and makes a cure in any case tedious, in some impossible.

[1] Case formerly reported by R. F. Weir.

Depending to some extent on the degree of constriction at the pylorus, the stomach is either hypertrophied or dilated. In infants an hypertrophy of the whole muscular structure of the stomach, associated not infrequently with a diminution in capacity, is more common than in adults. This condition is followed by dilatation, and it may be assumed that the primary hypertrophy is an effort to compensate for a narrowed pyloric lumen. Just what the factor is other than overwork which results in the secondary dilatation is yet unknown, and again the analogy with the clinical course of cardiac diseases may not be remote, since gastric dilatation like cardiac marks a loss of power to functionate. The hypertrophied stomach in adults may give rise to symptoms, especially when it is associated with anacidity, that will make a correct diagnosis very difficult. In Case VII the history of extreme loss in weight, together with the presence of an easily palpable tumor, and with patterns of abdominal tumidity over the stomach area, might well be interpreted as carcinoma. At the operation in this case the stomach wall was found much thickened, while the mucous membrane appeared quite normal. These instances of hypertrophy without dilatation as a sequel to the stenosis, however, are comparatively rare in adults, the common effect being a dilatation. In all of our cases, with the exception of Case VII, dilatation to some degree existed. The anatomical characteristics of this condition when it results from stenosis of the pylorus are in certain respects different from those of simple dilatation. In these cases the muscle layer of the stomach is usually as thick as normal and in the pyloric end it is often considerably increased in amount. The thinning of the gastric wall is usually due to thinning of the mucosa. This remains true although the dilatation resulting from benign obstructions is the most extreme that we encounter. Such marked grades of ectasy described by Bonet in the seventeenth century, in which the lower border of the stomach reached to the symphysis and the walls were likened to soft paper, are seen but seldom nowadays, because the general use of the stomach pump, as employed by Kussmaul in treating dilatation, has removed the cause of those extreme grades of the condition noted in the older literature. There are certain symptoms presented in these cases that are due primarily to motor insufficiency, of which fermentation is one of the most prominent. It is a symptom that always suggests stagnation. If the fermentation be of an acid nature, there will be pyrosis; if gaseous, the sensation of tension will occur shortly after eating. It has been held by some that fermentation does not proceed in the stomach when HCl is present, but this idea is in error. The contrary has been proved by Kuhn and Strauss. The evidences of fermentation were quite constant in our cases, usually it was gaseous, but in 2 cases lactic acid was found in the stomach contents.

A more or less marked change in the secretion of HCl is noted quite constantly, and it has been assumed that because a high degree of free HCl is a fairly common incident that it stands in causal relation to the spastic pylorus. The basis for this idea rests on the fact that in cases of acute pyloric spasm, in which there is frequently an accompanying increase in HCl, the symptoms are relieved by lavage. The conclusion has been drawn that we are dealing with cause and effect rather than with two results due to a third obscure factor. It is not always remembered that in acute stasis, products of fermentation are developed that might, and in fact do, produce as much irritation as a superabundance of HCl, which is a physiological secretion to which the musculature reacts only in a physiological manner. It is just possible that the increase in HCl is an effort toward compensation—in order that the thoroughly digested food may pass through the narrowed pyloric lumen when a less well-digested chyme could not. The cases wherein obstruction occurs through some agent outside of the pylorus, such as bands, adhesions, or enlarged glands, are so often marked by a hyperacidity, that the inference is strongly suggestive of this compensatory action. It is commonly accepted that the hypertrophied muscular coat is an effort of nature to force the food through its obstructed exit, and it is possible that the increase of acid may be a chemical compensation to the same end. The following cases of stenosis, due to extragastric causes, would seem to support this explanation of the hyperacidity:

CASE III.—Catharine B., aged forty-two years, was admitted to the New York Hospital complaining of stomach trouble. Her past history was unimportant. She says she drinks considerable coffee; occasionally a little whisky. Her present illness began five months ago, with discomfort in the abdomen after meals. This sensation she describes as one of oppression occurring an hour or so after taking food. She has vomited often at these times with immediate relief. The vomitus consists of food material, mucus, and froth. She states that she is often able to recognize in the vomitus, food that has been eaten the day previous. She has never vomited blood. She has much thirst, passes little urine and the bowels are constipated. She thinks she has lost considerable weight. Examination showed a dilated stomach and an excess of free HCl. At operation there was found a cirrhotic liver of the luetic type, and the pylorus was bound down by a fibrous band which passed around it. This band was divided and the patient recovered complete health.

CASE IV.—Patrick M., aged fifty years, complains of vomiting and loss of strength. His family history is negative. The patient has had no illness in his life except the present one. He has been a hard worker, moderately alcoholic; no history of lues. The present illness commenced three years ago with the regurgitation of sour, greenish fluid, eructations, and occasional vomiting. These symptoms have steadily increased in severity until a year ago, when he had to give up work. He vomits frequently at present, three or four times a week, and has a feeling of oppression in the abdomen almost constantly. The appetite is good and thirst increased. He frequently notices a splashing in his stomach after meals. Examination showed a dilated stomach containing a residue after fifteen hours fasting. There was a secretion of abnormal amounts of gastric juice on an empty stomach (gastrosuccorrhœa). The secretion of HCl was increased and the urine was scanty and alkaline in nature. The cause of the constriction was found at operation to be a narrow band passing around the serous surface of the pylorus. There was no sign of an ulcer. The band was divided at the operation, but the patient died of postoperative peritonitis.

Case V presented the same findings on examination as in the previous case, and gave the same history of gastric disturbances and vomiting. The HCl determinations varied between 40 and 80. The cause of the stenosis was found at operation to be adhesions between the pylorus and gall-bladder.

We need not enter into the cause of the anatomical conditions found in these 3 cases further than that the adhesions and constricting bands were not the lesions usually resulting from ulceration, and so of themselves throw no light on the cause of hyperacidity. The fact that a gastric condition identical in anatomy and chemistry to that caused by fibrous hyperplasia of the pyloric ring can be produced by pressure on the pylorus from without proves the falsity of the theory that the hyperplasia is a result of an hyperacidity. Whether the hyperacidity be a natural compensatory effort or whether it is only another manifestation of the ultimate etiological factor, cannot at present be decided. A solution which is satisfactory must also explain the excess of HCl which is a common occurrence in peptic ulcer.

The following histories are presented in a somewhat abridged form from the case books of the hospital:

Case VI.—Annie M. C., aged thirty-eight years. The patient says that she has had good health until ten years ago. She never had any acute illness. She has always been very nervous. For ten years she has had attacks of dragging pain in the left lumbar region, accompanied by the appearance of a firm, hard mass in the left hypochondrium. During these attacks she suffers acutely, and vomits frequently; the vomitus often contains food eaten the day before. At these times her skin always becomes "mottled." These periods of pain and vomiting have grown so frequent that she is now unfitted for her duties. The patient is a neurotic woman, poorly nourished. On the left side below the costal margin there is a tumor mass. The upper border is clearly felt, the lower border is not made out. The mass is smooth and somewhat mobile. The stomach is apparently dilated. The spleen is enlarged and movable. The patient was advised to submit to an operation, which was performed. Both kidneys were found movable, the left constituting the tumor mass felt. The spleen could be easily pulled down to the level of the umbilicus. Ptosis and dilatation of the stomach existed and the pylorus would not admit the end of the little finger; the ptosis produced a kinking that further narrowed the lumen. The patient was somewhat improved by efforts at fixation and pyloroplasty, but when last heard from in June, 1905, three years after operation, she was not cured.

Case VII.—Amelia C., aged twenty-seven years, married, was admitted November 2, 1903. The family history and the past history show nothing relative to the present trouble. Three years ago the patient began to have pain of a heavy dragging nature in the epigastrium and in the small of the back; made worse by taking food. This would continue for a few days and then subside. Except for these periods of pain, she was in good health until one year ago; she did not vomit and did not lose weight. During the past year these periods of pain in the epigastrium began to recur at shorter intervals, until they were nearly constant. She vomited almost all nourishment taken; the vomitus consisted of undigested food, but never blood. Her physician gave her lavage, which relieved the pain and vomiting. During the last six months she has been worse and has vomited more frequently, the pain being nearly constant. She has lost 75 to 80 pounds in weight. The patient is a very much emaciated young woman who looks anemic, but not cachectic. The abdomen presents peristaltic-like waves over the stomach area. Just above the umbilicus and to the left there is an indefinite mass about an inch in diameter, which is very tender and apparently movable with respiration. The mass feels smooth and not very hard. The stomach is not dilated. The liver and spleen are not felt. There is no glandular enlargement. The patient was in the hospital one week on the medical side and vomited every day save one, but never vomited blood. The vomitus was usually undigested food taken eight to fourteen hours before. An Ewald test breakfast was taken twice, giving the following duplicate result: Free HCl absent; total acid 50 and 70; lactic acid present; mucus present; no blood; no Boas-Oppler bacilli. In the lavage before the test meals particles of food taken the day before could be identified.

The patient did not do well on any diet and was transferred to the surgical division for operation to relieve the stenosis. At the operation the stomach walls were found thickened, but otherwise apparently normal; there were no masses in the wall. No signs of healed ulcer were found, but the pylorus was very hard and thick. A pyloroplasty was done. The patient was discharged December 19, 1903, improved. In March, 1904, fifteen months after the operation, the patient had gained about fifty pounds in weight and looked well. She said she had no trouble with the stomach and eats everything. In January, 1905, the patient says her health is perfect.

Case VIII.—Fred M., aged twenty-three years, was admitted January 2, 1902. Early history has no relation to present trouble. For the last fifteen years the patient says he has had attacks of pain and vomiting at irregular intervals. These attacks last three or four days and occur about once a month. He vomits undigested food and mucus. The vomiting has occurred of late more frequently and it is for this that he seeks relief. Examination was negative, except that the stomach was found to be dilated. Gastric analysis showed free HCl in excess; no blood. At the operation constriction of the pylorus without hypertrophy was found, with no signs of a former ulcer. The patient was lost track of.

Case IX.—Patrick J. McN., aged thirty-five years, clerk, was admitted March 10, 1902, complaining of pain in the abdomen. The patient has had neurasthenia and dyspepsia since January, 1902. There has been much pain over the stomach and nausea with gradually increasing vomiting. At the time of admission, patient was vomiting everything that was taken into the stomach; was emaciating rapidly and showed very positive evidence of obstruction of the gastric outlet and of starvation. He has not been out of his room since January, and has been in bed most of the time. He has been treated for lues. At the operation the pylorus was found unobstructed, but there was a large mass of glands behind and obstructing the duodenum. Gastro-enterostomy was done and a gland removed for examination. After operation the patient continued to feel badly and vomited three to ten times a day. He was discharged March 19, 1902, unimproved. The diagnosis was, obstruction of the duodenum from enlarged abdominal glands.

The gland showed a hyperplasia of small lymphocytes and of the larger cells of the sinuses, but the process did not appear to be at all malignant. The patient was put on a vigorous anti-syphilitic treatment and the symptoms all disappeared. The correctness of the ultimate diagnosis was substantiated by the later appearance of a superficial gumma of the leg which resolved under mercurial treatment. The diagnosis which led to the operation was based on the presence of a palpable tumor mass, the dilated stomach, constant nausea and vomiting, and emaciation. No gastric analysis had been made before admission to the hospital where he was sent for operation.

Case X.—John P., aged thirty-six years, a mechanic, was admitted February 1, 1903, complaining of vomiting and flatulence. The family and past history are negative. His habits are good. For several years the patient has had a little distress after meals, but this has not been serious and he had considered himself in good health until a year and a half ago. Then the distress became greater and he had much flatulence. The pain has never been acute and has been relieved by vomiting. The frequency of vomiting has varied, but since September, 1902, he has vomited two or three times a day. Vomitus consists of fluid and pieces of food eaten a day or so previously, but never of blood. His general health has become impaired and he has lost strength and flesh. Aside from the stomach symptoms the patient feels well. He is poorly nourished. The stomach can be seen occasionally bulging in a powerful peristaltic wave. The stomach border comes to the umbilicus.

An Ewald test meal resulted in the removal of a large amount of fluid which contained free HCl, 12; total acidity, 52. At the second Ewald test meal 200 c.c. was removed, containing orange pulp, eaten the morning before. Analysis showed free HCl, 30; and total acidity, 53. Lavage at various times revealed a motor insufficiency and an excessive secretion. As no course of treatment benefited the patient, he was subjected to an operation, at which there was found a thickening of the pylorus with narrowing of the lumen. Gastro-enterostomy was done. Eleven months after the operation (January, 1904) the patient was at work, feeling in perfect health. He had gained thirty pounds and was using no discrimination in diet, believing his digestion to be normal.

Case XI.—Amelia J. C., aged fifty years, a housewife, was admitted December 30, 1901. The family history was negative. Patient had smallpox in childhood, but no other acute sickness. Thinks she has been a very healthy woman, but has been nervous for thirty years. For several years beginning fifteen years ago, she had "hysterical" attacks, in which she would have convulsions and remain unconscious several hours, but she has had none of these attacks for ten years. Her present illness dates from August 1, 1901, when she received a severe fright. For several weeks after this fright she vomited every time she ate. The vomiting gradually ceased, but she has taken no solid food, living chiefly on milk and broths. She has no distress on taking food, but the thought of food nauseates her. She has lost twenty to thirty pounds. Physical examination is negative except for the stomach which seems to be enlarged. While in the medical ward, she vomited food taken three days before (prune skins and orange pulp). An Ewald test meal showed that free HCl was absent; lactic acid was present; there was no blood. The patient constantly lost weight and was transferred to the surgical division on January 27, 1902.

An exploratory operation showed a tumor mass the "size of a lemon" involving the pylorus and adherent to the under surface of the liver. A gastro-enterostomy was done. The patient recovered well from the operation and was discharged, February 25, 1902, with a diagnosis of carcinoma of the stomach.

After leaving the hospital she gained in strength, and her weight increased forty pounds. She was quite well and able to do her household work until July 20, 1903, when she again applied for admission to the wards. She says she had a fall three months ago and since then she has had pain in abdomen at times. The pain is confined to the hypochondriac region and does not radiate. She has also regurgitated food and had some distress after eating, but has not vomited. Her general health has not suffered; she has not lost flesh, but thinks she has gained during the summer.

Physical examination was the same as before. An Ewald test meal showed free HCl absent; total acid, 20; lactic acid, a trace; rennin and pepsin both absent; no pepsinogen. These results were confirmed by three test meals taken on alternate days. The patient improved on lavage and diet and was discharged August 3, 1903. The diagnosis at this time was chronic gastritis (achylia gastrica). She was re-admitted to the hospital on November 26, 1903. She has been gradually gaining strength since leaving the hospital and has had no pain or distress until five days ago, when the old pain returned and she vomited several times. She has vomited several times a day since, the vomitus consisting of greenish fluid. The vomiting holds no relation to the taking of food, but relieves the pain.

An Ewald test breakfast removed after one hour showed free HCl absent, and total acidity, 10. There was some mucus, some bile, no enzymes, and lactic acid was present. (Three test meals were made.) Lavage for the first three days after admission showed considerable bile, which later disappeared. The patient had one bad attack of pain in hospital, accompanied with vomiting of green fluid and fever of 104°. This all passed away in twelve hours. She was treated by lavage, low diet, and diluted HCl. She grew rapidly better and was discharged December 19, 1903. The discharge diagnosis was: Chronic atrophic gastritis and vicious circle. June, 1904: Patient has been well since discharge and has gained some flesh. She does her work at home and considers her health perfect. January, 1905: Health remains good. (Letter.)

This history covering four years makes the original diagnosis of cancer, in view of the patient's present health, very improbable. The case would seem to be one of true myoma of the pylorus, for the mass is still palpable in its original place and size. The present short attacks of vomiting of bile are doubtless due to a vicious circle having become established from the gastro-enterostomy operation. This symptom has caused no trouble during the last year, the patient's health remaining excellent.

A study of the symptoms of these 11 cases indicates the uncertainty of the diagnostic significance of two symptoms—gastrosuccorrhœa and achylia gastrica. The last case quoted (XI) has given a gastric analysis for many months of a typical achylia gastrica, and still the condition of the patient warrants in no wise an assumption of malignancy in the very evident pyloric tumor. In one other case of this series also, in which the stenosis was due to fibrous constriction, there was an absence of free HCl in the gastric analysis, although no test was made for the presence of pepsin. In other words, it is impossible to diagnosticate the presence of a malignant growth from the character of the gastric secretion alone, and it is in these cases that the tests for occult blood in gastric contents and feces are of most value. Gastrosuccorrhea also was noted in two of these cases; one of these was a typical fibrous stenosis and the other showed a narrowing due to the presence of an extragastric adhesion band. This combination of symptoms, the collection of a hyperacid gastric juice in the starving stomach, known as Reichmann's disease, has been described as occurring either in the stenosis due to ulcer or from the effect of neurotic influences. The two cases recorded here (IV and X) would extend this list to include benign stenosis among the etiological factors of a gastrosuccorrhea.

Successful medical treatment will be limited to those cases of benign stenosis in which the narrowing of the stomach outlet is developed only to a very moderate degree. It is not rational to expect to overcome a marked mechanical obstruction by a strictly medical procedure. In fact, it seems probable that those cases in which medical treatment has proved sufficient to control the periodicity of the symptoms, have presented not a well-marked stenosis but more the condition of a spasm of the muscular outlet of the stomach combined with a slight stenosis. There can be no doubt that the methods of treatment which can be used to relieve a spasm of the pylorus will often control for many years these milder cases and keep the patient in a comparatively good state of nutrition, although he will never be considered in robust health.

In the cases of intermediate degree of development also a great deal of time would be saved the patient if the condition could be recognized and proper surgical measures resorted to at an early stage.

In the cases of comparatively slight stenosis (or what amounts to the same thing during the early stages of a progressively narrowing pylorus), there is a distinct indication to use the means which we have, (1) to control excessive secretions of acid by the stomach; (2) to reduce pyloric irritability; and (3) to increase the muscular activity of the wall of the stomach. One or all of these medical procedures will tend to prolong the period of comparatively good health and to postpone serious surgical procedures perhaps indefinitely.

The method of medical treatment to control the secretion of acid includes the regulation of the diet to avoid irritating highly-spiced, richly-cooked, and also poorly-cooked foods. All articles which have been fried and which contain the results of the over-heating of fats must be eliminated. The diet must be regulated also to avoid the taking of large quantities of food at any one time, including the use of bulky liquid foods, such as soups and broths. The regulation of the taking of water so that it is ingested between meals and not mixed with the food is also important. The abstinence from irritating drinks which contain alcohol is necessary. The treatment would include also the use of gastric antispasmodics, among which one must especially emphasize belladonna, in doses of $\frac{1}{8}$ grain of the solid extract, given in pill form after meals. The systematic use of olive oil is beneficial when given in doses of from $\frac{1}{2}$ ounce to 2 ounces, usually a half hour before eating, as recommended in the treatment of gastric ulcer by Cohnheim. The combination of these two remedial measures, the use of olive oil before meals and of the belladonna after meals probably has a greater influence to control spasm of the pylorus than any other therapeutic measure, and at the same time, has a positive and distinct influence to overcome the natural tendency in these stomachs to hyperacidity.

A second important indication is to control the increased secretion of acid. In addition to this use of olive oil and belladonna, we can use astringents, particularly nitrate of silver by lavage, or less successfully in pill form. Or, we can use large doses of bismuth which seems to interfere mechanically with the normal irritation of food which is antecedent to the secretion of the acid. These last two drugs have been used particularly to influence the healing of a gastric ulcer, but they act undoubtedly in that condition not only upon the ulcer itself, but upon the hyperacid secretions as well. In addition to influencing the secretion of acid, one can overcome its effects by neutralizing the acid after it forms. Any of the alkalies are usful for this purpose, but perhaps those which do not give rise to the formation of carbonic-acid gas are better. The commonly used bicarbonate of sodium, therefore, should be discarded in favor of some of the alkaline salts and preparations of magnesium, particularly the oxide of magnesium. The magnesium preparations have also a distinct value in so far as they tend to overcome the sluggishness in the lower intestinal tract and cause a laxity of the bowels. The dose of the alkali to be used is more or less indeterminate, but a sufficient quantity should be taken to relieve pain and to neutralize the acid already formed.

The third measure of medical treatment consists in increasing the peristaltic action of the stomach walls. The one drug which is particularly useful for this purpose is ergot. The solid extract of ergot in grain doses can be combined with the belladonna already suggested and given in pill form after meals. The use of electricity has probably been of value for this same purpose and much has been claimed for it when used by the intragastric method.

The therapeutic value of the systematic use of the stomach tube is very certain, but it is difficult to control these patients in its use and they are likely to resort to the tube as soon as any discomfort follows the taking of food. In this way, they undoubtedly relieve themselves of their suffering, but they rob themselves of necessary nourishment and will often lose weight and strength more rapidly than if they are not taught this bad habit. On the other hand, there is no doubt that the stomach tube will often cure an attack of spasm, and its careful use will help to carry these patients for a number of months or even years in comparative comfort.

In these cases of pyloric stenosis which have existed for a considerable period of time we find, as has already been pointed out, that the mucous membrane of the stomach becomes atrophic and that the secretion of the normal ingredients of the gastric juice is more or less diminished. It is necessary, therefore, to consider in certain of these cases the means that we have at hand to restore the functions of a gastric mucous membrane which is the seat of an atrophic gastritis. The usual condition is a simple diminution or absence of the hydrochloric acid. This can either be supplied artificially or the mucous membrane can be excited to renew its secreting functions. It will frequently be found that some other mineral acid will be more easily borne than will the hydrochloric acid, and diluted nitric acid or the nitrohydrochloric acid will often give better results in these cases of gastric atrophy. In any case when it is necessary to prescribe an acid for this purpose, it is desirable that it be given in small, frequently repeated doses rather than in one large dose immediately after eating. A single drop of the dilute acid mixed with a dram of water should be given every twenty or thirty minutes for four or six times during the two hours immediately following each meal. The more usual method of prescribing ten or fifteen minims of acid, no matter how well diluted, immediately after eating, is very apt to cause gastric distress. The method of exciting an atrophic mucous membrane to resume its functions consists in the use either of stimulating drugs or of stimulating foods. Saline relishes such as salted fish, caviar, sardines, taken at the beginning of a meal will often have the desired effect. The use of peppers and other condiments such as mustard, horseradish, if not overdone, is also desirable. The drugs which act as gastric stimulants are the bitters, capsicum, and alkalies. Small doses of bicarbonate of sodium, with or without bitters or capsicum, are very useful, especially combined in a capsule and given before meals; either nux vomica or the simple bitters may be used for this purpose. Of the simple bitters, columbo and gentian are perhaps the most serviceable. They may be given as tinctures or as infusions. In those cases of anacidity in which the tinctures are not contra-indicated, the small amounts of alcohol will often prove beneficial. This line of treatment is particularly useful as a sequence to operative treatment in the cases of long standing.

When all has been said, however, in favor of medical treatment of pyloric stenosis, the final resort in cases of high degree of development must be to surgery. The operations which have been suggested to overcome the condition consist of two groups, one of which includes the several methods of enlarging the narrowed portion of the alimentary canal by the operation of Mikulicz or that of Finney. The second group includes the many forms of creating a false passage between the dependent portion of the stomach and a point in the intestine below the duodenojejunal junction. In referring any such patient to a surgeon for operation, the exact method of relief must be left to be selected at the time of operation and in accordance with the anatomical conditions found and the condition of the patient.

It might be well to record, however, some of the disadvantages of the two operations. The operation of pyloroplasty does not give as rapid relief as does that of gastro-enterostomy. The patients after these operations require careful watching and study to see that they do not return to a normal diet and normal method of living too rapidly. It will often take several months and even a year before such a patient has secured the full extent of benefit that is to follow the surgical procedure.

The operation of gastro-enterostomy on the other hand, sometimes fails to give relief by causing the condition known as vicious circle, which can only be cured by means of a second operation. The danger after this operation of a subsequent and complicating peptic ulcer in the small intestine below the anastomosis, is a real danger, although an infrequent occurrence, and the disarrangement of the coils of the gut resulting from this operation may rarely be the cause of a subsequent intestinal obstruction.

Both operations have their special indications, but we must confess to a very positive prejudice in favor of the pyloric operation, because of its undoubted approach to a nearer restoration of the normal condition.

In conclusion, we would lay stress upon the fact that the surgical treatment of the stomach has its most brilliant application in cases of benign stenosis of the pylorus and we would warn most strenuously against the too long postponement of surgical interference in cases in which medicine does not promptly cause a betterment in the symptoms, as evidenced by a longer interval between the attacks of obstruction and spasm and a permanent improvement in the general health of the patient. Surgery must not be kept as a last resort, but must be given a chance to cure these patients before the stomach has become dilated beyond recovery and before the mucous membrane has lost its power to return to the normal and again to secrete a normal gastric juice.

We would lay further emphasis upon the proposition that the mere correction of the mechanical error will not suffice for the cure of these cases. After the patient has recovered from the surgical procedure, he must be given the benefit of the methods of treatment which we possess to overcome the muscular atony of the stomach wall, to control the hypersecretion of HCl, or to excite the mucous membrane to resume its normal secretory function.

The result of treatment in the 11 cases which form the basis of this paper so far as known, may be tabulated as follows:

	Operation.	*Result.*
Fibrous stenosis, 6 cases	pyloroplasty, 4	2 cured. 1 partial cure. 1 unknown.
	gastro-enterostomy, 2	1 cure. 1 partial cure.
Fibrous bands, 3 cases	relief of obstruction, 3	1 cure. 1 died (peritonitis). 1 unknown.
Obstruction from glands	gastro-enterostomy, 1	cured.
Myoma of pylorus	gastro-enterostomy, 1	cured.

HEART CLOT IN PNEUMONIA.

By Beverley Robinson, M.D.,
of New York.

The question of cardiac thrombosis in pneumonia is still interesting and somewhat undetermined despite much work and very many observations. Clinicians and pathologists of former years admitted its frequency, as we know, and numerous deaths were attributed to it. Today pathologists, especially, recognize it as occurring very rarely, and, therefore, causing death only occasionally. Indeed, postmortem records of some large city hospitals (City Hospital and Bellevue of New York) among several hundreds of cases will not show a single instance in which the clot in the heart is particularly described, or any evident importance attached to it as being instrumental in bringing about a fatal termination.

Charles Norris,[1] pathologist to Bellevue Hospital, New York, states: "Since I have been in charge of the laboratory, beginning October, 1904, we have autopsied 130 cases of lobar pneumonia, and we have no records of a single case of cardiac thrombosis." Harlow Brooks[2] states: "In a series of 84 consecutive autopsies on my pneumonia patients, I have found but 3 cases of pulmonary embolism or thrombosis, and 1 of cardiac thrombosis. These cases do not, of course, include those cases where thrombosis was a generalized process, as it not infrequently is in truly septic cases." H. T. Brooks[3] writes me from the pathological laboratory of New York Post Graduate Hospital, that during the year 1905 "there were no records of cases of either cardiac or pulmonary thrombosis" in lobar pneumonia. I. Adler[4] writes that during 1905 (at the German Hospital, New York City) "we have had only 19 deaths. Only 8 autopsies are on record, in none of which the autopsy notes mention either cardiac or pulmonary thrombosis or embolism. In those cases that died and no autopsy was obtained, the clinical symptoms never pointed to cardiac or pulmonary thrombosis or embolism." John

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[1] Personal letter, June 22, 1906

[2] Personal letter, January 20, 1906.

[3] Personal letter, Febuary 14, 1906.

Personal letter, January 18, 1906.

S. Thacher[5] states: "We have not been able to find any records of cardiac thrombosis in acute lobar pneumonia during 1905." (Presbyterian Hospital, New York City.) "The firm clots found at autopsies often in pneumonia, were not recorded as the departure from the normal and its degree are so difficult to judge of."

On the other hand, F. C. Wood[6] affirms: "Clots which are formed in the heart are easily distinguished from those formed after death, by the fact that they are light in color and largely composed of fibrin and white corpuscles. In judging of the time of formation we have to consider whether the clot is adherent to the walls of the ventricles or auricles, and whether its texture is hard or soft." William H. Welch[7] writes: "Genuine antemortem clots are not common in the hearts of patients dead of pneumonia, although they occasionally occur. The age of cardiac thrombi can be determined with some degree of probability by careful examination." And yet clinical observers, even now, do not always share these beliefs. Several to whom I have spoken or written state frankly that they believe antemortem clots in pneumonia occur many times in fatal cases, and are much to be dreaded. Henry Jackson,[8] of Boston, Massachusetts, writes: "I do think that antemortem heart clot is a frequent cause of death" in penumonia, "as suggested at times by sudden death when the general condition does not seem to warrant it."

Bond Stow states[9] "cardiac thrombosis in cases of pneumonia is a very interesting and important subject." S. S. Cohen[10] is also of that opinion, as the following shows: "I am aware that modern pathologists discard the old theories of death from antemortem clot, but admitting the alterations in the constitution of the blood and realizing the fact that the circulation in the pulmonary artery and its branches is obstructed, delayed, and in some places brought to a standstill, the mechano-biologic conditions are present for the formation of thrombi and the suddenness with which death sometimes occurs in patients whose other symptoms have given no intimation of the imminence of cardiac, vascular, or respiratory failure, or of general exhaustion, seems to point to the actuality of such occurrences. Measures to maintain the fluidity of the blood are therefore wise."

H. A. Hare[11] believes that thrombosis in croupous pneumonia "is very much more common than Osler thinks."

A. C. Morgan[12] states: "Cardiac clot is said to occur frequently in fatal cases, but whether this is the cause of death or part of the closing scene is not settled."[13] The late Austin Flint, as we know, was a firm believer in the existence of antemortem heart clot as a frequent cause of death in pneumonia. Certainly, in view of the greatly increased amount of fibrin factors in the blood of pneumonia patients, these clinical statements are, at least, much to be relied on, and yet it is only fair to add that "venous thrombosis" is an exceedingly rare complication of pneumonia. Steiner[14] could find only 38 cases recorded, and reports "three of his own." Why then believe, it may be argued, in the frequency of cardiac thrombosis which is no more probable? How is this difference of opinion to be explained? From the pathological point of view, antemortem clots which have formed a few days preceding death, terminal clots, or those which are formed during the agony do not differ materially and always from clots which unquestionably are formed after death. In both instances they may be largely formed of red-blood globules and present simply the appearance of current jelly. Or again, they may have the aspect of chicken fat throughout a portion of the coagulum and the rest of the clot be a mixture of red blood and fibrin with a predominance of the former. In neither case are they very intimately associated with the trabeculæ of the heart, or at all attached to the heart wall with any degree of firmness. The clots are not of themselves hard, nor do they show any appreciable stratification. We do find, however, in pneumonia, and quite as often, coagula, which are almost entirely fibrinous, do adhere intimately to the heart wall, and are bound up with, and intimately attached to, heart valves. These clots may be, and usually are, distinctly firmer than the others, and now and then show separate layers which could not be formed at the same time. When these fibrinous clots are present they have commonly the print of the aortic or pulmonary cusps obviously shown upon that portion of the clot which extends into the aorta or the pulmonary artery. Sometimes, indeed, "discoid masses formed usually almost exclusively of fibrin, fill the cavities of the cusps and are moulded to their surface. To this, great importance has been attached as indicating the formation of the coagula prior to death. In fact, Poullet has endeavored to prove by experiments upon animals, that in all cases in which these masses were present the clot had been formed quite a length of time during life."[15]

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[5] Personal letter, May 8, 1906.
[6] Personal letter, June 9, 1906.
[7] Personal letter, June 2, 1906.
[8] Personal letter, June 5, 1906.
[9] Personal letter June 28, 1906,.
[10] Jour. Amer. Med. Assoc., December 10, 1904.
[11] Personal letter, December 18, 1905.
[12] New York Med. Jour., October 13, 1906, p. 743.
[13] It should be borne in mind that acute dilatation of the heart is sometimes the cause of death in pneumonia among adults, and even among infants, in which at the autopsy no antemortem clot is found. See Medical News, June 3, 1905, p. 1032.

These facts indicate clearly to me that such clots may be found before death. Moreover, this judgment has been experimentally confirmed, as stated, on animals during life, and if analogy has value must prove equally true as regards man. The pathologists state that this is not admissible because such clots occur in diseases other than pneumonia, although less frequently, and when they do occur they give rise to no special signs or symptoms, and if that be true, How can it be otherwise in pneumonia?

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[14] Hare's Practice, 1905, p. 161; also see original paper in Johns Hopkins Hosp. Bull., 1902, p. 130.

[15] Pepper's System of Medicine, vol. iii, p. 730 and 737. Formerly, I was inclined to believe that reasonable doubt might be cast upon Poullet's experiments. At present, I am disposed to sustain their validity.

Clinicians answer that these clots may occur in other diseases, notably in diphtheria, but not as often as in pneumonia, and when they occur, owing to the gravity of the disease, with many and alarming symptoms due to pulmonary and other structural changes and also to the profound toxemia, they are not clearly made out. Even in diphtheria, whenever fibrinous clots are found in the heart, in the great number of cases, there is a complicating pneumonia present, and this pneumonia, undoubtedly, is an additional cause of heart clot. There are, moreover, cases of pneumonia in which the grave symptoms which suddenly arise, seem to point to cardiac coagula, and the rapidly fatal termination of the disease shows it almost unerringly. The heart clot may occur during the acute stage of pneumonia, when other symptoms are grave; it may occur just after the crisis, when everything appears hopeful; and it may occur during the convalescence period, when all danger is seemingly passed.

In connection with the heart clot there usually is pulmonary thrombosis, and very exceptionally pulmonary embolism. The latter condition is likely to be present when the embolic plug has come from a vein of the lower limbs (saphenous) and been carried through the right heart.

Unfortunately, in many cases in which death has occurred rapidly or suddenly and in which it is fair to assume that heart clot is present, no autopsy is permitted. This is notably true in private practice. Again, when we have made an autopsy, we find structural changes in the cardiac muscle which to many appear the direct cause of death, and the heart clot merely a result of these changes and of no particular importance. Here, again, I am inclined to believe such statements are misleading, because the heart changes are not, in my judgment, sufficient to cause death. They are no more, probably, than what we find in other cases of pneumonia in which death has not occurred, or in instances in which the death is apparently not due to the condition of the heart, if we can fairly estimate the condition of lungs, kidneys, and nervous system. Likewise, in other acute diseases, and notably typhoid fever, the intramuscular heart changes are greater than in pneumonia and do not cause death so often as it occurs from the heart in pneumonia. I am convinced that in a healthy individual attacked with pneumonia, without previous heart or kidney disorder, the heart will usually hold out sufficiently to preserve life during the acute stage and later, if the blood is kept as far as possible in a condition approximating the normal, namely, if the chlorides are properly replenished and saline beverages taken in such quantity as to keep up diuresis. (Todd.)

Cohen,[16] also argues that "the disturbance in chloride excretion seems to point to the wisdom of increasing the sodium chloride content of the blood."

H. S. Carter, on the other hand, thinks that the role the chlorides in the blood play is small, but fully believes that anything to promote diuresis by large quantities of fluids, saline or otherwise, by mouth, bowel, or hypodermoclysis "is of the greatest value." "We thought," writes Carter, "at first that as some of the cases showed rather an increase of chlorides in the blood, we might be able to graduate the percentage of saline solutions given, with a view to bring about a normal average, but as the percentage of blood chlorides, even in the severe cases did not average high, that idea was of little value. What a large series might show I cannot say. However, of 16 cases, 12 were fatal, so that they should have shown it more regularly, if it were always or even usually an accompaniment of a serious or fatal infection. I think, to be on the safe side, in severe cases I should depend much more on the diuretic effect of the fluid itself rather than the percentage of saline in it, and I would give a saline solution of rather less than that which is isotonic with normal blood, 0.9 per cent. I do feel that the nitrogenous products are much the most important to combat, of chemical constituents; that is shown well by this small series depending more on the amount of lung involvement than the apparent severity of the infection. The toxemia, of course, is always better for dilution, not, however, by intravenous use of the saline, so much as by the other more natural routes."[17]

Of course, if simple endocarditis be present, we could have a condition which would render clotting probable, even though the blood be in relatively good condition. Fortunately, simple acute endocarditis in pneumonia is infrequent. On the other hand, when it occurs, it does so comparatively early, and unfortunately "the diagnosis is always difficult and often impossible."[18]

To have a good physiological basis for any remedial practice which appeals to us, is always desirable if we can obtain it. In the question of heart clot in pneumonia, I have found nothing which in this direction is more than doubtful, but that much at least permits honest empirical trials. "It is not believed that there is a normal definite chemical in the blood which prevents clotting in the vessels in the normal state. The present view is that the "fibrin ferment" is formed first when blood comes in contact with anything except the lining of the vessels, and that perhaps even the formation of said ferment is due to something else, which is formed first, probably, out of disintegrating blood plates, or leukocytes, or both. Once formed thus, the ferment converts a proteid of the blood plasma, fibrinogen, into fibrin." Why, inasmuch as blood plates and leukocytes disintegrate "in the system" in health, they do not, by liberating ferment, cause clotting—nobody knows. Whatever they give rise to may be at once consumed by the cells of the vascular wall, or these cells, or some cells, may give out something unknown which may neutralize the nascent ferment."[19] From a pathological point of view we have to do less with mere conjecture.

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[16] Jour. Amer. Med. Assoc., December 10, 1904.

[17] Personal letter, September 25, 1906.

[18] Edward F. Wells, Jour. Amer. Med. Assoc., October 18, 1902.

Intense reaction in pneumonia consists of cellular metabolism, which tends to reduce the alkalinity of the blood. The reduction of alkaline salts in the blood brings a tendency to precipitation of fibrin, and increase of toxins entails cardiac arrest. With the continuance of pneumonia the alkaline salts are more and more reduced and are not replaced by the ordinary restricted dietary, hence the vital defences are continuously diminished, and finally life cases.

When a proper specific gravity and alkalinity of the blood are maintained, the crisis of the disease is precipitated by the continuance of a high temperature which tends to destroy the pneumococcus. With this view of biology, Todd[20] first treated pneumonia patients with salines. The views of Todd are also held essentially by F. P. Henry, and by Cohen. The latter believes in them particularly, because he thinks that the pneumotoxin creates a marked tendency to "coagulative processes." Todd prescribes from the inception of the disease 2½ grains of sodium chloride and 1 grain of potassium bicarbonate to an ounce of water, and 6 to 8 ounces of this fluid should be taken every two hours. The addition of lemon juice makes the drink more palatable. The character and quantity of the urine is the best test of the efficacy of the treatment. If in large quantity and light, it shows the specific gravity of the blood is normal; a different condition of the urine, viz., when high colored and in small quantity, indicates that more alkaline drink should be taken.

John B. Todd[21] writes: "My experience this past winter has confirmed my belief in the usefulness of the saline beverages in pneumonia. I believe the early and continuous use of salines does prevent, in a measure, the tendency to fibrin formation; it washes out the toxin and keeps the blood and tissue serum to maximum quantity, and in that way favors the leukocytosis, and at the same time the diuresis tends to temperature reduction." J. Madison Taylor,[22] in the "Symposium on Pneumonia" before the Medical Association of the Greater City of New York, December 11, 1905, practically endorsed the views of Todd. I commend it as a further and valuable contribution to "the role of saline solution in the treatment of pneumonia."

It is probable that the addition of lemon juice to the saline beverages is really useful in view of the researches of Professor Wright, who has found that the giving of citric acid decalcifies the blood and thus lessens its tendency to coagulation. According to W. Haward, C. J. N. Longridge has made similar observations. Haward[23] thinks, therefore, that citric acid should certainly be given as a curative measure when thrombosis has occurred, and also as a preventive agent in all those instances in which this complication is likely to occur. Wright[24] and Haward[25] believe that 20 to 40 grains of sodium citrate to every pint of milk is indicated, because it produces a partial decalcification of the milk and thus restricts the intake of lime salts. It also renders the milk more digestible. This it does by lessening the amount and density of the curd.[26] A similar amount of citrate of sodium may be added to every pint of saline solution when administered by the rectum. If increase of coagulability of the blood depends upon excess of lime salts in the blood, and not upon the increase in the albuminous elements of the fibrin, Wright's[27] view appears eminently reasonable. We know, indeed, that "cows' milk contains 1 part in 600 of CaO as compared with 1 part in 800 contained in lime-water."

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[19] Personal letter from John G. Curtis, March 8, 1906.

[20] New York Med. Jour., March 20, 1905, p. 1004.

[21] Personal letter, April 17, 1906.

[22] Medical Record, January 13, 1906.

In view of the foregoing it seems that we should give citric acid in the form of lemon juice, properly diluted and slightly sweetened,[28] far more frequently than we do at present, not merely as an agreeable, refrigerant drink, but also for its notable action in preventing coagulability, whenever there is reason to anticipate or dread it, and this is especially true in all cases of pneumonia.

On the other hand, Augustus Wadsworth[29] writes: "The calcium salts are only part of the subtle equilibrium by which the coagulability of the blood is controlled. Furthermore, the relationship of the coagulability of the blood to the infection and the immunity of the patient, is a field of research not yet explored with any practical results. The French observers, Gilbert and Fournier, "consider the fibrinogenesis in pneumonia, in some subtle way, an all-important factor in bringing about the recovery from the infection. These observers might, therefore, question the advisability of using citrate infusions[30] in the great majority of the cases."

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[23] Lancet, March 17, 1906, p. 741.

[24] Medico-Chirug. Trans., 1903, vol. lxxxvi. [25] Loc. cit.

[26] Wynn, Birmingham Med. Review, April, 1906. Poynton and Shaw, Archives of Pediatrics, March, 1906, also corroborate this view.

[27] Wright has suggested, also, that a possible cause for the frequency of phlebitis as a complication of typhoid fever lies in the increased coagulability of the blood caused by cows milk. Therap. Gaz., May 15, 1906, p. 315.

[28] Sugar is stated to prevent coagulation. Vide Article Coeur, Dict. de Med., vol. viii.

[29] Personal letter, July 26, 1906.

[30] This letter of Wadsworth was in reply to my asking whether it would not be desirable, in certain urgent cases, to add citrate of sodium to the ordinary saline venous infusions. I had in mind, also, other conditions in which cardiac thrombosis might occur—notably diphtheria, typhoid fever, and following profound surgical shock from accident or operation. I recognize in this latter condition, with B. H. Caswell (Boston Med. and Surg. Jour., June 28, 1906), that salt solution, when alone, given intravenously, is of little or no benefit as it does not raise the blood pressure. To effect this, adrenalin is the only drug to be used. The proportions of adrenalin are: 1 to 1000 solution, one part in 200,000 (?) parts of saline solution, or 1 dram to the pint. (Hare.) I am glad to note that H. A. Hare holds the same views as Caswell. Vide Practice of Medicine.

The tendency to coagulability of the blood is also diminished, as we know, by oxygen inhalations, alcohol, and diminution in the quantity of food taken. As regards oxygen, we may say "the better the oxygenation of the blood, the more easily it circulates . . . Moreover, the more thoroughly the blood is oxygenated, the less prone is it to coagulation. It has been proved that the inhalation of oxygen gas diminishes the coagulability of the blood."[31]

As the result of Bence's observations, in a large number of cases, he finds that "the viscosity of the blood rises and falls with the quantity of carbonic acid which it contains. . . . By reason of this increased viscosity, blood overloaded with carbonic acid naturally exerts its effect on the heart. If the overloading is the result of cardiac insufficiency, the latter defect will be further increased. In suitable cases, inhalations of oxygen will reduce the viscosity of the blood which contains abnormal quantities of carbonic acid, by favoring the elimination of the latter, and in this manner the cardiac insufficiency may likewise be relieved."[32]

We all know the views of Benjamin Ward Richardson with respect to the effect produced by giving ammonia internally. He states that it made coagulability feeble and slow and also made the blood globules irregular. Bartholow has, also, the belief that ammonia has considerable value in preventing and curing cardiac thrombosis.[33] In a letter from Paul Bartholow, April 15, 1906, he writes me as follows: "My father had had a great deal of experience with the effects of ammonia in those cases where the blood serum was altered by those rather indefinite bodies which we now believe factors in the phenomena of agglutination. In practice, I have twice recently seen good effects from the aromatic spirit of ammonia, which by the way has been recommended by Osler."[34]

S. S. Cohen,[35] also, states: "Ammonium, too, is known to oppose the deposit of fibrin. In the absence of an efficient antitoxin, the old empiric use of ammonium chloride and ammonium carbonate and the modern use of saline infusion seem to have their philosophical justification."[36]

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[31] Haward, loc. cit., p. 744.

[32] Med. Record, February 24, 1906, p 305; also, Zeit f. klin. Med., vol. lviii, No. 3. Article by J. Bence.

[33] Therapeutics, twelfth edition, p. 237.

[34] See the chapter on Heart Disease in his Practice.

[35] Jour. Amer. Med Assoc., December 10, 1904.

[36] Flint writes as follows: "I have for several years advised the carbonate or muriate of ammonia during the progress of the disease, with a view to prevent cardiac thrombosis. Of course it is difficult to obtain clinical proof of the protective efficacy of this or any other remedy against that accident. There can be no objection to the use of this remedy on therapeutical grounds, since it does not in any manner affect unfavorably the progress of the disease. A. Patton, of Indiana, in a paper advocating the free use of this remedy throughout the disease, that is from 5 to 10 grains every three hours, states that of 96 cases in which this constituted all the medical treatment, in only 2 cases was the disease fatal. AMER. JOUR. MED. SCI., October, 1870. Of 207 cases treated chiefly with this remedy, by J. P. Thomas, only 3 were fatal. (Vide Va. Med. Monthly, April, 1880.) "It is probable that digitalis given with a view to increase the power of the heart's action is useful in preventing heart clot." (Flint.)

On the other hand, W. H. Howell, of Johns Hopkins University, writes to me March 26, 1906, as follows: "In regard to the action of ammonia, I do not recall that anyone has seriously believed that ammonia can effect the clotting of the blood, since the old experiments of Richardson in 1856. Richardson abandoned his theory, I believe; certainly it has long since dropped out of physiology."

In a paper read before the Climatological Association,[37] June 4, 1904, I wrote as follows: "I am of the opinion that there is weighty evidence to prove that the solution of ammonia is a preparation, used moderately and continuously in pneumonia, of considerable value. Its value is specially shown not as a stimulant, because in that particular I believe alcohol, as whiskey or brandy, to be more desirable, but to keep the blood alkaline and fluid. In this disease, especially in the grave forms and where there is extensive pulmonary consolidation, it is to my mind a very important matter. I have seen many patients in whom, before death and at the autopsy, I have been convinced that heart clot avoided would possibly or probably have meant a life saved. To no one as much as to Benjamin Ward Richardson is the honor due of great insistence upon this important fact, too much ignored today in medical writings and medical practice.[38]

"In some instances where there was distention of the right heart and jugulars, with great breathlessness, pallor, or almost lividity of lips, face, extremities, and even portions of the trunk, which means *beginning thrombosis*, I have seen temporary good effects produced by a venesection, and occasionally, perhaps, a life saved.

"But many times reducing the bulk of blood will not save life unless we dilute it, and incidentally, perhaps, partially neutralize its contained toxins. Give, therefore, a moderate saline infusion after the venesection, with this hope, and also insist upon beginning immediately or continuing frequently the use of ammonia under form of the strong solution or the aromatic spirit."[39]

Finally, I would cite from a paper of mine read before the Association of American Physicians in 1894: "In one instance which I saw with J. E. Traub, of New York, and in which we bled the patient about 17 or 18 ounces at the third day of the disease, we had at first notable relief to the breathing and circulation, but soon the symptoms of distress and restlessness came back in aggravated form, and the patient grew suddenly worse and died. In this case no murmur could be heard in the cardiac region, and although the sounds were slightly muffled and the action extremely rapid, we only suspected the formation of heart clot, without being able to affirm it. In view of the sudden death of the patient and without other sufficient causes satisfactory to explain it, I now believe cardiac thrombosis was the immediate cause. I have not infrequently, especially in former years, and when I was a hospital interne, seen many autopsies in which pneumonia undoubtedly occasioned the heart clot which was the direct cause of death."

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[37] Amer. Jour. Med. Sci., December, 1904.

[38] I am glad to say that the late Austin Flint shared my views. Vide Practice, sixth edition, p. 172.

[39] The views here expressed are more affirmative than those I stated in an article entitled "Cardiac Thrombosis" which I wrote for Pepper's System of Medicine, in 1885, vol. iii, pp. 745 and 746. In this article, indeed, H. A. Hare, personal letter, December 18, 1905, finds a statement indicating that I have little faith in this means of preventing this occurrence. Later and wider experience has evidently changed my views considerably.

In those cases in which cardiac thrombosis is threatened, nitroglycerin is frequently advised. If there be high arterial tension associated with a rapid-acting heart, which is extremely rare, this is good counsel. If there is evident vasomotor weakness and cardiac failure is suspected, I agree with Le Fevre[40] in strongly condemning the tendency of resorting to it, as "peripheral resistance is already too low on the arterial side, and nitroglycerin only makes it lower."

Ewart writes[41] emphatically that he opposes the use of calcium salts "in a disease" (pneumonia) "so prone to clottings that one of its common fatal terminations is intracardiac thrombosis" (p. 778). Further, he strongly advocates a defibrinating treatment to forestall consolidation. He regards "clotting in the alveoli" as "the danger special to pneumonia." The aim, writes Ewart, should be at an *early date* "to maintain in the congested areas as much fluidity of blood and lymph as possible, to prevent vascular and lymphatic obstruction, and thereby to promote a *steady rate of elimination of the infecting agents and of their toxins*, thus avoiding or lessening the possible disaster consequent upon their concentration." Ewart advises the *use of leeches*, "of withholding fibrin formers from the fever diet, and of administering the citrate and the iodide of potassium to reduce the viscosity of the blood."

In conclusion, I would affirm:

1. Heart clot as an immediate cause of death in pneumonia is more frequent than postmortem investigations, as now made, apparently sustain.
2. Clinical experience seems to justify the statement that the carbonate of ammonium has probably considerable efficacy, when properly given, in preventing heart clot.

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[40] Some Problems in the Treatment of Pneumonia. Med. Record, February 24, 1906.

[41] "On the Use of Calcium Salts as Cardiac Tonics in Pneumonia and Heart Disease." London Lancet, March 30, 1907, pp. 778 and 779.

THE INTERPRETATION OF X-RAY PICTURES AS AN AID TO THE EARLY DIAGNOSIS OF THORACIC ANEURYSM.

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Of all the accusing ghosts of the medical conscience, aneurysm claims a place well toward the front rank. This stricture is based upon the evidence that: (1) The condition is frequent;[1] (2) the diagnosis is seldom made until the disease is practically unmanageable; (3) the condition is preventable; (4) it is curable; and (5) the chance of cure diminishes very fast as the size of the tumor increases.

Even in this day of diagnostic dexterity it is not an uncommon occurrence for medical men, who may be admirably skilled in most directions, to carry on their visiting lists patients who are steadily going to ground with unrecognized aneurysm. The cough excited by the condition is not infrequently misinterpreted, and practitioners in Colorado sometimes find such patients to have been sent hither because of a supposed affection of the lungs.

It is not intended to give here a *resume* of the symptomatology of aneurysm. No one can deny that the student whose conception of the condition is founded solely upon the classical description of the disorder will rarely make a correct diagnosis until the anomaly has reached proportions which menace life itself.

Professor Osler[2] has recently summarized his vast experience in this field and fixed upon the subjective symptom of pain as the earliest common manifestation of aneurysm. We venture to follow the chain of events a little farther back and fix upon the signs of local increased intrathoracic pressure as the earliest recognizable evidence of aneurysm. This pressure may cause no abnormal sensation; it may be manifested only by unusual dyspnœa with muscular exertion; it often, by partial occlusion of a bronchus, diminishes the intensity of voice and breath sounds in a lobe of the lung without modification of percussion resonance; it so commonly irritates laryngeal nerve branches that any interference with the normal mobility of the vocal cords, or even a cough which is not explainable by obvious causes, should lead at once to the suspicion of unphysiological intrathoracic pressure.

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[1] According to the United States Census Bulletin, the number of deaths from aneurysm in the United States in 1900 was 751, being 0.8 in 1000 deaths. In 1890 the rate per 1000 was 0.6. The only vital statistics of Denver which are definitely appropriate to this subject are recorded for the years 1895 to 1898, inclusive. During this period there were noted 13 deaths from aneurysm, including 1 from "aortic disease." This gives a ratio of over 1.86 deaths from aneurysm to 1000 deaths from all causes in Denver for the four years. Whether this larger proportion is due to greater diagnostic acumen on the part of our medical fraternity, or to climatic conditions, or to habits which superinduce the pathological state, is not clear. It seems probable that the number of deaths ascribed to aortic aneurysm is everywhere below the truth.

[2] Medical Chronicle, Abst. in Med. Rev. of Reviews, Oct. 25, 1906.

From the considerable list of causes that may lead to such abnormal pressure, the selection of aneurysm can only be made after further analysis. It cannot be too impressively stated that aortic aneurysms of considerable size may be completely latent as regards the production of signs and symptoms which are usually held to be necessary for diagnosis. And yet we venture to assert that should the medical observer bring into this field but a fraction of the technique, judgment, and intuition he employs in the study of abdominal disorders, aneurysms would usually be discovered in their incipiency.

The first essential to the early diagnosis of aortic dilatation is a psychological state. The observer must never forget the possibility of aneurysm being a cause of symptoms emanating from the chest; and when there is a suspicion of syphilis in the history of the patient, we believe with Saathoff[3] that rigid inquiry should be instituted to exclude arterial degeneration. It is rare indeed that the patient, seen early in his disease, presents signs of aneurysm distinctive from those of pressure that may be caused by a solid tumor mass. The Röntgen rays, the skiagram, which should be tested by the fluoroscopic findings, should be directed to every case of doubtful nature. But to the beginner in the study of Röntgen pictures the chances of error in diagnosis are thereby profoundly increased. The interpretation of such images by the untrained eye is about as reliable as would be inferences founded on the sounds heard through a stethoscope by an untrained ear. Sailer and Pfahler,[4] in a valuable contribution, say, "The investigators who employed the fluoroscope or skiagrams for the purpose of studying changes in the aorta were early impressed with the fact that, although aneurysm was occasionally overlooked, it was more frequently diagnosticated when it did not exist."

The chief inherent difficulty in the interpretation of the skiagram probably depends upon the fact that the shadows give little or no perception of depth. The observer must learn to draw inferences from two dimensions only without artificial device to simulate perspective. Probably every clinician who has sought the aid of the Röntgen rays in his diagnosis can recall examples of errors of judgment immeasurably increased by misinterpretation of the pictures. Such an instance has already been reported by one of us.[5] It may be useful to recount another case or two occurring in the practice of the same observer:

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[3] Das Aorten-aneurysma auf syphilitischer Gründlage und seine Frühdiagnose. Münch. med. Woch., liii, Nr. 42. Abst. in Jour. Amer. Med. Assoc., 1906, 46, 2129.

[4] Tortuosity of the Aorta, Amer. Jour. Med. Sci., Oct., 1903.

[5] Sewall, An Extraordinary Case of Anthracosis Simulating Aortic Aneurysm, Amer. Jour. Med. Sci., Jan., 1906.

CASE I.—A man, aged thirty-seven years, a smelter hand, for the last two months of his active life was employed in a smelter for mercurial ores. He positively denied venereal infection. His height was considerably over 6 feet, and his weight in health 240 pounds. Signs of slight mercurialization appeared while in his last position; his physician declared that mercury had settled in his liver and caused enlargement. His complaint was of extreme weakness. Feet and legs became swollen, and he suffered from shortness of breath. He manifested orthopnœa for two months previous to entering the Denver County Hospital, October, 1904. He had lost about 20 to 40 pounds in weight in six months. On examination the patient appeared to be in good general condition; his only complaint was of dyspnœa, especially marked with exertion. There were the physical signs of double hydrothorax, the effusion being larger on the left side. The heart sounds were practically normal, but very faint and distant. There was no "Broadbent sign" of pericardial adhesion. Each pleural cavity was tapped at intervals and clear serum was withdrawn; not more than about one quart could be obtained at one sitting. The negative character of the patient's history and the lack of positive disorder in his heart and kidneys made him a subject of especial interest. It was concluded that his hydrothorax depended upon pressure on veins entering the heart and that the cause of obstruction was probably a mediastinal growth. On one occasion a trocar, inserted into the chest near the left posterior axillary line, about an inch above the angle of the scapula, when it had penetrated less than three inches struck against what was apparently a hard, solid object. Here, then, there appeared to be proof positive of a new-growth within the chest. The patient was well enough to go to the office of Dr. G. H. Stover, one of the radiographers to the hospital, for *x*-ray examination. The skiagram showed a dark shadow rising from the surface of the fluid remaining in the left pleural cavity, apparently just in the situation it should have occupied if caused by a mass as located by the trocar. The diagnosis of a solid growth, probably of mediastinal sarcoma, was now considered settled. In the course of two or three months the patient died under other medical care, and an autopsy was obtained. The postmortem examination failed to reveal anything in the nature of a tumor. The left pleura had become adherent throughout and had a thickness of about one-quarter inch. The "solid body" struck by the trocar during attempt at paracentesis was undoubtedly the sheet of pleura which had been pushed before the point of the instrument until its resistance became palpable. The pericardium was very thick and densely adherent all over the heart. The clinical history and the fatal ending are easily explained by the postmortem findings.

Case II.—A man, twenty-eight years of age, came to Denver on account of faintly marked tuberculous infiltration at the apices of his lungs. Syphilis was denied. After a sojourn of a month it was evident that a fresh invasion of the pleura covering the upper lobe of the left lung had occurred, the morbid signs being especially marked along the left sternal border. At the same time a pulsation synchronous with the heart beat, and having an outward systolic excursion, appeared in the first left intercostal space within an inch of the sternum; there also developed a slight but distinct tracheal tug. A skiagram of the patient's chest, the plate having been placed on the back, showed a well-marked shadow above the heart, left of the vertebral column, as if made by a considerable dilatation of the descending aorta. Seen through the fluoroscope, the outlines of the shadow pulsated with the heart beat. The dimensions of the image were considerably less in a skiagram obtained with the sensitive plate situated on the front of the chest; the object in question was, therefore, nearer the anterior than the posterior wall of the thorax. When the abnormal shadow was viewed through the fluoroscope it diminished in diameter and practically disappeared when the patient, in the upright position, turned his body so that the rays penetrating the thorax should cut at a certain angle the anteroposterior plane. The interpretation of the shadow then became clear; it was simply due to the opacity of the pleura of the left lung, seen in optical section, bounding the anterior mediastinum. When the incident rays fell obliquely upon the sheet of thickened tissue the shadow was broad; when the rays were parallel to the membrane the shadow narrowed. The various pulsations and the tracheal twitch were also easily explained as the result of pleuropericardial adhesions.[6]

Lest the use of the Röntgen rays should seem to introduce more difficulties in diagnosis than it dissolves, it may be well to cite two more cases in which the radiographic pictures proved of vital therapeutic assistance:

Case III.—A gentleman, aged forty-seven years, who had had syphilis in his youth, was an active business man, carrying great responsibilities. His weight was about 240 pounds, his musculature rather flabby. He smoked to excess and took several drinks of strong spirits after office hours. He developed a dry, hacking cough, which was exaggerated with excitement or exertion. Physical examination of the patient revealed nothing noteworthy, except a diminution in the intensity of the respiratory murmur, voice sounds, and tactile fremitus at the left apex, while the percussion resonance was unchanged. An *x*-ray picture of the chest revealed a slight but obvious bulging of the aortic arch upward and to each side. The patient was cut off from his liquor, his indulgence in tobacco was limited, his physical exertions were greatly reduced, his diet was lightened, and iodides were administered in small doses. In a few days improvement was manifest and before long all morbid symptoms disappeared. Another *x*-ray picture taken some eight months after the first showed a much more normal aortic shadow.

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[6] In an essay by one of us (Sewall, Some Conditions Other than Aortic Aneurysm which Determine the Occurrence of the Tracheal Tug, Trans. Assoc. Amer. Phys., 1901, xvi) it was pointed out that pleural adhesions of the left apex were prone to give rise to a slight "tracheal tug." The case cited above would indicate that adhesions involving the mediastinal pleuræ would tend to produce the same phenomenon.

Case IV.—A lady, aged fifty-five years, suffering from chronic pulmonary tuberculosis marked by very slight infiltration of the left apex, but by a dry cavity at the right apex, suddenly developed a dry, spasmodic cough and hoarseness with use of the voice, and there appeared a marked pulsation, synchronous with the heart beat, in the first left costal interspace, about one inch from the sternum, and in this area there was a marked systolic bruit. The larynx was normal except that the left vocal cord was found partially paralyzed and in nearly the cadaveric position. This patient gave a history of having had five miscarriages. Moreover, for ten days preceding the sudden development of the symptoms described she had rested, for a purpose that need not be described here, with the foot of her bed elevated about seven inches above the floor. The skiagram showed slight globular enlargement of the extremity of the aortic arch with extension upward, a condition which without the clinical symptoms could readily be ascribed to simple "tortuosity" of the aorta. But when we consider the suspicion of luetic infection aroused by the history of five miscarriages there is ground for assuming arterial degeneration. Again, Stephens[7] has found that the maximal arterial blood pressure is very much increased in the "head down" position. In a study of 22 healthy young men Dr. Stephens found the means of his estimation of blood pressure, measured in the left arm, to be, standing, 130 mm. Hg.; sitting, 130; supine, 148.3; head down (body at an angle of 45 degrees) 165; right lateral, 114; left lateral, 156; It is not now necessary to consider the discrepancies between these results and those found by other observers, notably by Erlanger and Hooker[8] in their admirable treatise.

If Stephens' conclusions be true, we have an explanation of the sudden development of pressure symptoms; for nothing is more common in the history of aortic aneurysm than the sudden development of symptoms following exertions that raise arterial pressure. Moreover, the skiagram of this patient showed a marked enlargement and density of the bronchial glands, the presence of which could easily have opposed to the aorta a counter-resistance capable of generating pressure symptoms which would otherwise be wanting.[9] It may be said that during a rest in bed for nearly five months all symptoms attributable to aneurysm gradually disappeared.

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[7] Blood Pressure and Pulse Rate as Influenced by Different Positions of the Body, Jour. Amer. Med. Assoc., Oct. 1, 1904.

[8] An Experimental Study of Blood Pressure and of Pulse Pressure in Man, Johns Hopkins Hospital Reports, vol. xii, 1904.

When it was first discovered that the body could be made transparent, the Röntgen rays were supposed, necessarily, to offer a plain revelation of every anatomical secret. When it was found that mistakes in diagnosis might be multiplied and intensified by the general application of the method, there was a widespread reaction of discouragement as to its value. There is evidently still need of general information as to the limitations and conditions of usefulness of the *x*-rays in diagnosis.

We have been so strongly impressed with the difficulties of correctly interpreting the shadows of the thoracic skiagram that we venture to believe that a categorical summarization of the elements of confusion that have developed in our experience may be of service to beginners in this important field of diagnosis, when the *x*-rays are employed to ascertain whether thoracic aneurysm exists. Our conclusions are similar to those of Baetjer,[10] whose valuable paper was not seen until our work was finished:

1. In the study of fluoroscopic and skiagraphic pictures the beginner is apt to forget the fundamental importance of centring the rays and maintaining them as nearly as possible perpendicular to the plate or screen. Even when anode and screen or plate are so far separated that the incident rays are approximately parallel, a slight inclination of the receiving surface distorts the outline of the image, just as an object on the ground throws a distorted shadow in the light of the setting sun. No proper comparison can be made of the opposite borders of a Röntgen shadow unless the corresponding rays fall upon the sensitive plate at the same angle. Very gross errors as to the size and shape of the objects within the chest, as, for example, the mediastinal pleura or the arch of the aorta, may arise from failure to dwell upon this physical fact.

In a skiagram obtained with the central rays perpendicular to the chest at the lower border of the manubrium, the plate being at the back of the subject and the tube at least twenty inches from the plate, the top of the normal aortic arch should appear to be about opposite the fourth costal interspace behind.

2. It must never be forgotten that the *x*-ray picture usually

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[9] One of us has notes on two other cases, one having come to autopsy, in which extraordinary clinical symptoms, notably paroxysmal vomiting, seem properly to be ascribed to irritation of vagus nerve branches pinched between an intermittently dilated aorta and a much enlarged bronchial gland. Dr. Robert Levy, of Denver, whose laryngological observation of tuberculous persons has been very extensive, in a personal communication states that some five or six cases of laryngeal paralysis, due apparently to pressure upon a recurrent laryngeal nerve, have come under his care. The disorder usually involves the left vocal cord. In two patients, at least, the function has been restored. The paralysis was ascribed to the pressure of enlarged bronchial glands; such bodies were prominent in skiagrams taken in two cases.

[10] X-rays in the Diagnosis of Aortic Aneurysms, Johns Hopkins Hosp. Bull., January, 1906.

forms but one link in the diagnostic chain. The significance of the skiagraphic findings can often only be inferred after careful consideration of the clinical history, signs, and symptoms furnished by the patient. A wide range of anomalies of the thoracic aorta and its branches is to be found in the literature. Moreover, a condition well described by Sailer and Pfahler as "Tortuosity of the Aorta" is so commonly found in healthy subjects, as well as those manifesting vascular senile changes, that it must always be eliminated in considering the significance of a broadened shadow of the aortic arch. These authors point out that, especially in atheromatous subjects, the aortic arch may be elongated and its unsupported parts may be considerably displaced, as is so frequently seen in the peripheral arteries in conditions affecting their elasticity. The clinical determination of atheroma of the aorta is not without difficulty.

As pointed out by Klotz,[11] arteriosclerosis is apt to invade by preference vessels in the parts which are most used. It has been held that hypertrophy of the heart accompanies sclerosis of the aorta, but not sclerosis which involves peripheral arteries alone. The "tortuosity" of the aorta gives a skiagraphic picture which might easily be mistaken for aneurysm. But, owing to the topographical relations of the aorta, it is more common for the bulge in the shadow to occur to the left, at the descending part of the arch, than to the right. But, unless there is a true dilatation of the vessel, the image of the transverse portion of the arch is not likely to rise above its normal position. Our own experience has been chiefly with younger subjects than those studied by Sailer and Pfahler, but it confirms their contention that spurious bulging of the aortic shadow, particularly at the descending part of the arch, is a common phenomenon, and that it is especially likely to be found where arterial elasticity is diminished. We have not tested the matter thoroughly, but we venture to think that the fluoroscopic study of the patient while, in the sitting position, he turns round a vertical axis, should aid in the discrimination of elongation from dilatation of the aorta; with mere elongation of the artery the bulging of the arch should diminish when the rays become more nearly parallel to the plane of the arch. The conditions of the observation are, however, rather complex, and each case must be judged by itself. In our experience, bulging of the shadow to the right of the vertebral column is much more indicative of a pathological condition than an extension to the left. Extension of the aortic shadow upward, especially if associated with a bulge to the right, is very suspicious of a pathological condition.

3. Dilatations of the aortic arch, particularly in its ascending limb, are of frequent occurrence and must always be discounted in

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[11] The Relations of Experimental Arterial Disease in Animals to Arteriosclerosis in Man, Jour. Exper. Med., Aug. 1, 1906, p. 504.

the subjects of atheromatous vessels. It is of the highest importance, from a clinical point of view, to determine whether such dilatations are potential aneurysms. In our opinion, a history of syphilis in such a patient is sufficient ground to justify the medical observer in instituting treatment appropriate to the cure of aneurysm, as already advised by Saathof. Nevertheless, here, too, physical and circumstantial evidences may apparently conspire in leading the observer to wrong conclusions, as witnessed by the following case:

Case V.—Mr. G., aged twenty-six years, twelve years ago contracted syphilis, for which he was treated for about two years. He has had pulmonary tuberculosis about five years. For three years he has suffered much from chilly feelings in springtime. No history of malaria. Physical examination: Weight, 117 pounds. Wheezing rales throughout lungs, especially in right upper lobe, where there are obscure cavernous signs above and below clavicle. Air enters lower right lobe poorly. The apex beat is in fifth space under the nipple, but also faintly felt in sixth space, parasternal line. The area of "aortic dulness" is much enlarged, including the upper sternum and an area more than an inch broad on each side of it. The aortic second sound is somewhat accentuated. There is a slight tracheal tug. In the second right interspace, under the middle of the clavicle and to the right of the aortic dulness, is a pulsation outward in systole, both palpable and visible. In the second left interspace, close to the sternal margin, is a very small area of pulsation inward at systole. The veins of upper extremities are somewhat prominent, especially those of right arm and upper right chest. The abdominal veins are prominent in the erect position. There is noticeable epigastric pulsation, which is outward in systole on left border of epigastrium. There is very slight outward systolic heave of upper right chest. Lying supine, there is considerable pulsation above the clavicle, but nothing can be felt. There are no bruits; the radial arteries are slightly thickened. The pupils are equal and the pulses of even volume. There is no chest pain or significant cough.

When viewed by the Röntgen rays this man's chest presented typical difficulties in interpretation. The aortic arch was slightly higher than usual, and there was a perceptible outward bulge to its descending portion. Opposite this point, in a position corresponding to the ascending aorta, the shadow broadened in exact imitation of a dilatation of the artery. A dense band of tissue passed from the heart base, tangent to the apparent aortic shadow, upward and outward, to be lost in the apex of the right lung. Through the fluoroscope this band was seen to have a plain respiratory excursion, and it bounded the lower border of a small tuberculous cavity under the right clavicle. The pulsation in the second right interspace probably had its origin in adhesions of this band. Two skiagrams and a fluoroscopic study of these relations convinced us that the apparent dilatation of the ascending aorta, in spite of the clinical signs suggesting it, was spurious and due to tuberculous infiltration and distortion of the right mediastinal pleura and adjacent structures. Incidentally, the skiagram revealed a well-developed cervical rib on the right and a rudimentary one on the left of the patient's neck.

When repeated skiagraphic studies of a doubtful case through long intervals of time becomes a routine practice, disaster to patients from the insidious development of arterial degeneration will doubtless often be averted.

4. Relatively little notice is given by technical writers on this subject to confusion arising from the configuration of the bronchi and pulmonary bloodvessels. The shadows of these channels are often lost in the development of the plate. In a skiagram taken as described above, the bronchial tree and pulmonary artery in each lung are commonly represented by a single, short, horizontal trunk projecting from the spinal shadow on each side at about the level of the seventh dorsal vertebra, and dividing at a distance of about two inches from the middle line into two branches, one of which ascends parallel to the spinal column and the other descends in a direction somewhat obliquely outward.

Radiographers seem not to be in accord in the interpretation of these shadows.[12] Some take them to be the outlines of the bronchial tubes, while others hold that they represent the pulmonic arteries. Theoretically, it ought to be easy to distinguish the shadows of an air-filled tube and a blood-distended vessel; the one should increase in density toward the middle and the other on the margins. In the subjects of peribronchial sclerosis and adenitis the bronchial tree undoubtedly forms the basis for a considerable part of the figures in question. Enlargement, calcification, or pigmentation of the groups of lymph nodes at and near the bifurcation of the trachea is pretty sure to be represented in the trunk of the bronchial tree springing from the spine at the level of the seventh dorsal vertebra.

In normal lungs without undue development of connective tissue or enlargement of the lymph nodes, it is improbable that the course of the dividing bronchial tubes is to be traced in the radiogram. But it can easily be proved that figures corresponding in position to the outlines of the "bronchial tree" can be developed at will by overfilling the pulmonary bloodvessels. Our demonstration of this fact was founded upon and modified from an ingenious experiment of Professors Zuntz and Loewy and their collaborators,[13] who studied the changes in heart volume under extreme differences in the phases of respiration. Our purpose was to compare the skiagrams of a normal chest taken (1) when emptied as thoroughly as possible of blood; (2) when congested as fully as might be. In the first case the subject, an adult male with spare figure and normal lungs, at the end of an ordinary expiration, closed the mouth and nostrils and then strongly contracted all the muscles of expiration during the period of exposure. In the second case the subject, at the end of an ordinary expiration, closed mouth and nose and then attempted to draw air into the lungs by contracting the muscles of inspiration. The relative anemia or congestion of the lungs was additionally furthered by taking the skiagrams with the subject supine, in the first case with the body inclined, head up, at an angle of about 12 degrees to the horizontal, and in the second case with the head down at the same inclination.

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[12] P. M. Hickey, The Interpretation of Radiographs of the Chest (and discussion), Trans. Amer. Rontgen Ray Society, 1905, p. 136.

[13] Hohenklima u. Bergwanderungen, 1906, S. 352.

Detailed description of the pictures so obtained will be given in another place, but it is pertinent to state here that the skiagram of the congested lungs shows well-marked figures corresponding to the ramifications of the "bronchial tree" which are absent from the companion plate.

When the system of peribronchial glands is well developed, particularly if there is a condition of pneumonokoniosis, the outlines of the bronchial tree, with the attendant arteries, can lead to error, particularly through the simulation of an aneurysm of the descending aorta by the tubes supplying the lower lobes of the lung.

5. In any disorder of the lungs attended by sclerosis, as is so often witnessed in pulmonary tuberculosis, the heart is likely to be drawn far out of its normal position. We have especially noticed that displacement of the heart to the right tends to develop a perceptible tracheal tug. The shadow of the displaced heart made by the *x*-rays may lead to confusion as to the meaning of the images about it. In such cases there is likely to be a greatly thickened pleura and enlargement of the bronchial glands, which combine to broaden the shadow from the base of the heart upward.

6. A tumor of the œsophagus could with difficulty, if at all, be skiagraphically differentiated from a saccular swelling of the aorta.

7. The mediastinal glands, particularly in tuberculous subjects, are a fruitful source of error in diagnosis. Moreover, the chains of infiltrated glands in the lower thorax can easily suggest a fusiform dilatation of the descending aorta. A group of enlarged glands at the aortic arch may, according to our observations, give a massive shadow, pulsatile under the fluoroscope, which it is exceedingly difficult to differentiate from an aneurysm. The medley of enlarged glands, thickened pleura, and cirrhotic tissue enclosing pulmonary cavities may cast a shadow almost exquisite in its imitation of aneurysmal dilatation. In such cases it is usually easy to avoid error by noting the homogeneity and regular outline of the aneurysmal shadow as compared with the unequal density of surface and more or

less knobbed outline of its spurious imitation. As already hinted, clinical experience has strongly impressed us with the suspicion that the presence of enlarged glands about the aortic arch is peculiarly likely to result in the manifestation of nerve-pressure symptoms, with very slight degrees of arterial dilatation. A nerve trunk might be widely displaced by a swelling artery without provoking signs of irritation; but if the sensory cord is pinched between the vessel wall and an unyielding body, such as an enlarged, hardened gland, the slightest abnormal dilatation of the artery might be expected to provoke profound sensory disturbances. Our experence indicates that certain cases of angina pectoris and of paroxysmal vomiting may have their origin in this way.

8. Pneumonokoniosis may lead to the precipitation of dense shadows throughout nearly the whole or any part of the thoracic skiagram, and can easily be so situated as to simulate saccular aneurysms. A mistake in diagnosis made by one of us in this direction has already been referred to.

9. The thickened pleura is remarkably opaque to the Röntgen rays. It is frequently the case, especially in the subjects of pulmonary tuberculosis, that in the development of the *x*-ray plate the image of the heart is completely obliterated before the shadow formed by the obscuring pleura can be dissolved, especially when the plate is placed at the back. An actual error in diagnosis, largely due to the conformation of a thickened pleura, has already been recorded above. We have been especially impressed with the importance of the mediastinal pleuræ as occasionally giving rise to suspicion of aneurysm of the descending aorta. The apparently expansile pulsation which the heart movement may give to these membranes is very suggestive of a vascular tumor. Before drawing conclusions from the radiographic pictures in respect to these sources of error, there should be a comparison of skiagrams obtained with the sensitive plate upon the front of the chest with that made in the usual manner. By reason of the divergence of the *x*-rays a given object will appear larger and less well defined the farther it is situated from the sensitive plate and the nearer it is to the source of the rays. It is particularly serviceable to analyze the fluoroscopic image by viewing it along different radii of a horizontal plane, as can be easily accomplished when the patient, sitting upright, turns his body on a vertical axis. By this means, for example, the dense shadow thrown by a pleural membrane when penetrated obliquely by the rays may be seen to contract its borders as the sheet of tissue is viewed edgewise.

THE RELATIVE VALUE OF HIGH AND OF LOW ALTITUDE IN THE TREATMENT OF TUBERCULOSIS.[1]

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While I recognize that our ideas are more or less influenced by our surroundings, and that men who practice at high altitudes are likely to favor it in treatment, while those practising at low altitudes are likely to think it best, yet we must divorce ourselves from such influence and discuss the question in an impartial manner, if we wish to arrive at the truth. The fact that one observer sees tuberculous patients do well at a high altitude does not prove that high altitude is the most advantageous place to treat tuberculosis; nor does the fact that another observer sees tuberculous patients do well at low elevations prove that such elevations are the best for tuberculous patients. These observations simply point to the fact that some tuberculous patients do well at high, and others at low, elevations; or, as it might be stated broadly, patients suffering from tuberculosis can get well at various altitudes. It remains, then, for us to discuss impartially, if such is possible, the conditions accompanying both high and low altitude, and then attempt to suit these conditions to the patient and his disease.

A few years ago it was thought that there was a specific climate for tuberculosis. Individuals suffering from the disease were led to believe that if they could only leave home and go to one of these favored places they could be cured. And they also felt that if they were to remain at home it was almost certain death. So strongly has this idea taken hold of the people that they are led to do many foolish things. They leave home without any idea of where they are going or what they are going to do when they arrive at their destination. The thing that they have uppermost in their minds is reaching this favorable clime. All else is secondary. But the time comes when the "favored climate" is secondary, and meeting the conditions of life and adapting themselves to the climatic change is primary. These difficulties and annoyances could be largely eliminated by carefully advising patients before they leave home.

The task of choosing a climate for an individual suffering from a disease is one of great importance, for unless it is properly suited it is capable of doing harm. The idea is prevalent that anyone can make a change of climate. So they can, but it is not everyone that can make an intelligent change. To prescribe climate requires as much skill as to prescribe drugs. In prescribing a climate for one suffering from tuberculosis the disease and the individual both must be kept in mind.

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[1] Read at the twenty-fourth annual meeting of the American Climatological Association, Washington, D. C., May 7, 8, and 9, 1907.

The principles underlying climatotherapy are not so difficult, if only one stops long enough to understand them. The beneficial effect of a change upon nutrition is well known, even if the change be one only of surroundings, and not of climatic conditions. The effect of going to a less favorable climate than that in which one lives is often beneficial. How much more beneficial, then, should a change be which is carefully suited to the patient and his disease.

The primary effect of climatic treatment is upon metabolism. Its fundamental effect is upon tissue change, and its effect depends upon its powers of abstracting heat. The heat abstracting power of a climate depends upon the temperature, humidity, and the movements of the air. A cold climate, then, with much wind, makes the greatest demands upon the system for heat; a warm, humid climate, with absence of wind, should make the least. A dry, warm climate cannot be equable, because it does not hold heat well, and as soon as the sunshine ceases, or as soon as the shade is entered, the air becomes cool. The wide diurnal variation in dry climates is due to this fact.

Thus it can be seen that a climate which makes great demands for tissue change requires a hardy man to endure it. It would be unwise to send one who has low vitality, and who endures cold badly, to such a climate with the expectation of his deriving benefit from the change, for his forces could be overtaxed in supplying the normal waste incident to such rigorous conditions. On the other hand, such a patient would doubtless improve in strength and weight very materially if surrounded by such climatic conditions as would allow of the body cells keeping up the natural tissue change and still having some power in reserve to apply to the building-up of a stronger constitution. Cold climates are stimulating, while warm, humid climates are enervating. Between these we have all grades, and so, if we form a correct opinion of our patient, we can find a suitable climate for him.

It must be remembered that climatic conditions change with the seasons. Thus the climate of the Rocky Mountain regions at an altitude of 5000 feet is cold and rigorous in the winter, but temperate in the summer. Many people who might not be able to endure the winter might profitably spend the summer months there.

One suited to life in a cold rigorous climate, or at high altitude, should belong to the hardier type of men, with good circulatory, digestive, and assimilative powers. The young and robust are particularly suited to such conditions, while those in advancing years should be spared the excessive demand for tissue change.

The first point, then, that should be considered in choosing a climate for a tuberculous patient is his ability to respond to the demand for tissue change, which will be made by such climatic change. This point must be determined upon regardless of the question as to the adaptability of high altitude to the treatment of tuberculosis.

For many years high altitude was considered to be an essential in the treatment of tuberculosis. This opinion was due largely to the statements of Dr. Archibald Smith, who wrote, in 1840, that in the Peruvian Andes immunity from tuberculosis was commonly observed at an elevation of 7500 to 8500 feet. This was very evident, because in the cities at lower altitudes and at the base of the mountains tuberculosis was rife. This same apparent immunity was noted in the Rocky Mountains and also in the Alps. The natural conclusion is that high altitude confers immunity. This was honestly believed for many years.

Other observations, however, were made, which cast some doubt upon the supposed immunity being due to the altitude per se. It was found that the Steppes of Tartary, some portions of which are below sea level, and the great desert of the Southwest in America, portions of which are also below sea level, likewise share this apparent immunity from tuberculosis. This forced the conclusion that it is not altitude per se, but certain conditions which exist in common between high altitudes and these great desert regions which causes this apparent immunity.

If we stop to inquire what these conditions are, we note that both have: (1) A sparse population; (2) a great amount of sunshine; and (3) a pure air with low bacterial content.

When we consider that these are the conditions which are favorable to the prevention of the spread of tuberculosis, we can readily explain why tuberculosis does not exist to any extent in such regions. Tuberculosis is a disease which is most rife where overcrowding exists. It is particularly a disease of cities, and especially prevalent among the poor. Sunlight is the greatest enemy of the bacilli and our most reliable prophylactic, consequently we would not expect tuberculosis to spread very rapidly in these sparsely populated districts.

The votaries of high altitudes have endeavored to explain its benefits as being due to things which are not in common with low altitudes, and they have almost invariably used the damp lowland climates in the comparison. Consequently, they claim that the value of high altitude is due to lowered atmospheric pressure, a greater diathermacy, an increased electric tension, dryness of the atmosphere, and a great amount of sunshine. Granting that these are the elements of value, they are all present, except the first, in the low altitudes possessing dryness of atmosphere; consequently, there must be some other reason assigned to their supposed superiority of high altitude or we must accept the conclusion that high altitude is not an essential to the treatment of tuberculosis.

Now let us examine the other side of the ledger and see if there are any reasons why high altitude is contra-indicated in the treatment of tuberculosis? In the first place, high altitude treatment is contrary to the principle of rest in inflammations in general and tuberculosis in particular. The accepted principle of treatment in tuberculosis is rest, and it is employed whenever possible, so it is well to stop and inquire whether or not we should make an exception in case of the lung, and, if so, why so?

That high altitude causes great activity on the part of the lungs and throws great strain on the heart which is already severely taxed is an accepted fact, and one which cannot be ignored in the discussion of this subject.

That rest is the better method of treatment of pulmonary tuberculosis is also suggested by the observation which I have made repeatedly with reference to that portion of the lung which immediately surrounds the heart. When this part becomes seriously affected, especially on the left side, where it receives the greatest impulse of the heart beat, it is very slow at healing. I believe that the proper explanation of this fact is associated with the constant motion of the part owing to the action of the heart.

Another point relative to the employment of high altitude is the condition of the patient. It seems unreasonable to place an individual who is deprived of a portion of his aërating surface under conditions where he is subjected to a natural demand for more respiratory effort, and one whose heart is undergoing an extra strain under circumstances where it will be subjected to more strain. It also seems unreasonable to place one who is suffering from the severe bodily drain incident to tuberculosis, whose vitality is lowered, whose bodily functions are interfered with, in such conditions as to be called upon for an exaggerated tissue change, such as is required to meet the demand which is made by a high altitude. This consideration calls in question the advisability of sending those who have advanced lesions to a high altitude, and suggests that if a high altitude is the best place for treating tuberculosis, only those who have slight lesions should be chosen for such treatment. Further consideration would also suggest another important limitation, that is, of early cases only those should be sent who are able to meet the extra demand made upon them by the climatic conditions found.

Another important point in the treatment of tuberculosis is the after-result. It has long been noted that patients who secure an arrestment of their trouble at high altitude must remain there afterward. It seems to me that the explanation of this might be suggested by another observation which is common to men who see many cases of tuberculosis, that is, that tuberculosis is common among athletes. It seems to me that the reason for this lies in the overstraining. Athletes are subjected to more or less constant strain. Their heart and lungs over-develop to meet the extra burden thrown upon them.

They become larger than is necessary to carry on the usual work associated with the ordinary duties of life. When they leave the athletic field and settle down to the usual routine of life, they are at a disadvantage. They have more lungs and heart than they need. Nature attempts an adjustment. A retrograde process supervenes, the organs become more or less flabby and less resistant. That this is probable we may infer from our observations on muscles which have been over-developed and those subjected to disease.

When one becomes adjusted to a high altitude the heart and lungs increase in size just as those of the athlete, and when he returns to the low altitude his heart and lungs must undergo a readjustment, a retrograde process must take place. In the case of tuberculosis it is probable that during this time the tissues are not so resistant as usual and that the patient is prone to have renewed activity in the old process.

From these observations I am led to the conclusion that tuberculosis is a disease which is best treated at low elevations.

THE RELATIONS BETWEEN DIABETES AND PREGNANCY:

WITH THE REPORT OF A CASE OF DIABETES IN WHICH THE GLYCOSURIA DISAPPEARED WITH THE INCEPTION OF PREGNANCY AND REAPPEARED AFTER DELIVERY.[1]

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Diabetes mellitus must be looked upon as the expression of a derangement of carbohydrate metabolism, resulting in the more or less constant presence of an excessive amount of sugar in the blood, and, secondarily, in the urine, together with profound nutritional disturbances. The transitory appearance of sugar in the urine is not rarely observed in conjunction with a number of other conditions, while occasionally in cases of diabetes the sugar disappears from the urine with the onset of acid intoxication and coma, and perhaps it may be absent also at other times.

Sugar has been observed so frequently in the urine of pregnant, parturient, and puerperal women that its presence has been designated physiological. H. Blot[2] found in the urine of all parturient and nursing women and in that of about half of all pregnant women a reducing substance that he considered sugar, and he thought the phenomenon related directly to the secretion of milk.

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[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 7, 8, and 9, 1907.

[2] Gaz. d. hôp. civiles et milit., 1856, p. 482.

Leconte[3] contested this view, contending that the reducing substance was not sugar, but rather uric acid. T. Kirsten,[4] on the other hand, agreed with Blot, while E. Brücke[5] maintained that normal urine almost constantly contains traces of sugar and that the urine of puerperal women may contain even larger amounts.[6]

A. Hempel[7] concludes that sugar appears in the urine of puerperal women only as the secretion of milk becomes active, and that the amount is proportionate to the degree of development of the mammary glands and the duration of the stasis of the secretion in the breasts. V. Johannovsky[8] made comparative observations on 25 healthy men and 25 nursing women and found that the urine of all of the former and of 21 of the latter (without milk stasis) reduced copper and was levorotary, while the urine of 4 nursing women with milk stasis likewise reduced copper, but was dextrorotary. He considers the latter phenomenon as evidence of a resorption lactosuria, and L. Hofmeister[9] found the reducing substance actually to be lactose.

J. Ney[10] examined the urine of 148 nursing and 24 pregnant women and found sugar present in four-fifths of the former and one-sixth of the latter. The condition was especially associated with an abundant secretion of good breast milk. G. Keim[11] found sugar in the urine of 20 among 25 nursing women and in that of but 4 among 19 pregnant women. He considered the condition physiological in the one instance and pathological in the other. Among 125 women between the seventh and ninth months of pregnancy M. Brocard[12] found sugar in the urine in 50 per cent. Glucose predominated, but milk sugar appeared as lactation was approached.

F. A. Lemaire[13] found that when sugar was present in the urine prior to labor the amount became greater after labor in consequence of the excretion of milk sugar, as well as of a greater amount of glucose and isomaltose. Milk sugar appeared in the urine of nursing women when none had been present during pregnancy.

F. Lanz[14] has found the power of assimilating carbohydrates greatly diminished in pregnant women, as shown by the presence in 19 of 30 cases from the fourth month of pregnancy onward of grape sugar in the urine within six hours after the administration of 100 grams of grape sugar. R. v. Jaksch[15] thought this phenomenon might be diagnostic of the existence of pregnancy. J. Hofbauer[16] found the assimilative power for carbohydrates progressively reduced in 39 of 45 cases from the end of the second month of pregnancy, and he considered the reaction as indicative of the existence of physiological conditions.

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[3] Compt. rend. hebdom. des séances de l'Académie des Sciences, 1857, xliv, 1331

[4] Monatsschr. f. Geburtsh. und Frauenheilk, 1857, ix, 437.

[5] Sitzungsbericht der kaiserlichen Academie der Wissenschaften, Wien., 1858, xxviii, xxix.

[6] Wien. med. Woch., May 8, 15, 1858, 321.

[7] Archiv f. Gynak., 1875, viii, 312.

[8] Ibid., 1877, xii, 448.

[9] Zeitschr. f. physiolog. Chemie, 1877–78, i. B, 101.

[10] Archiv f. Gynak., 1889, xxxv, 235.

[11] Progrès médical, 1898, i, 424.

[12] Compt.-rend. hebdom. des séances et mémoires de la Société de Biologie, 1898, t. 5, 10 ser. 1077.

[13] Hoppe-Seyler's Zeitschr. f. physiolog. Chemie, 1895–96, xxi, 442.

[14] Wien. med. Presse, December 8, 1895, xxxvi, Jahrg., No. 49.

H. Ludwig[17] contends that the organism of a healthy pregnant woman reacts upon polysaccharids in the same way as does that of a non-pregnant individual, so that a failure to demonstrate diminished assimilation of carbohydrates cannot be accepted as evidence of the non-existence of pregnancy, while the appearance in the urine of a pregnant woman with abundant glycosuria of more than 1 per cent. of the sugar ingested with a mixed diet is of affirmative diagnostic significance. He examined the urine of 82 women for mellituria during the last eleven weeks of pregnancy and found no trace of sugar in 26, fermentable sugar alone in traces or in amounts determinable quantitatively in 10, fermentable sugar in traces or in amounts determinable quantitatively, but not alone, in 38, and varying amounts of fermentable and non-fermentable sugar in 8. A. Payer[18] determined that the assimilative power for grape sugar in pregnant women is in inverse relation to the maturity of the fœtus; that is, the farther advanced the pregnancy the more readily can alimentary glycosuria be induced. Other things being equal, alimentary glycosuria can be more readily induced in primiparæ than in multiparæ. The disparity is more marked with advancing years. On the other hand, Payer found glycosuria uncommon during pregnancy.

J. Veit[19] suggests that the glycosuria of pregnancy may be due to absorption of the glycogen contained in the chorionic tissues. He observed dextrose in the urine of rabbits when living young placental tissue was introduced into the abdomen.

In the course of 1800 urinalyses Robert N. Willson[20] noted that when sugar appeared in the urine of a pregnant woman not previously glycosuric the phenomenon was observed between the beginning and the end of the last month of gravidity. Occasionally a trace of glucose was observed throughout pregnancy, often disappearing completely after parturition.

Although sugar is thus said to be a frequent constituent of the urine during pregnancy, diabetes, on the other hand, appears to be quite an uncommon complication of gravidity. This fact is attributable to the circumstance that the disease is less common in women generally than in men, and that its incidence in women is greatest after the child-bearing period. There are, however, on record a small number of cases in which diabetes has recurred with successive pregnancies. Even rarer than diabetes complicating pregnancy is the occurrence of pregnancy in a diabetic woman. The reasons for this, in addition to those already given, are to be found in the state of impaired nutrition of the diabetic patient and the functional derangement and structural alteration of the internal generative organs that are often present.

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[15] Prag. med. Woch., July 4, 1895, xxvii, 281.

[16] Wien. klin. Rundschau, xii, Jahrg., January 1, 1899, 1.

[17] Wien. klin. Woch., March 23, 1899, xii, 12, 305.

[18] Monat. f. Geburtsh. und Gynäk., 1899, x, 559, 784.

[19] Münch. med. Woch., July 17, 1906, 1437.

[20] Amer. Jour. Med. Sci., 1904, cxxvii, 261.

J. M. Duncan[21] quotes Frerichs as stating in a private communication that of 386 cases of diabetes mellitus 282 occurred in males and 104 in females, and that of the latter only 1 was observed during pregnancy. From personal experience and a study of the literature Duncan concludes that diabetes may develop during pregnancy and even be present only in association with that condition; or that it may disappear with the termination of pregnancy, either to appear some time later or not to recur in a subsequent pregnancy; or that it may develop soon after labor. He recognizes the fact also that pregnancy may occur in a diabetic woman and, with parturition, be unaffected by the associated disease, although liable to be interrupted in its course by death of the fetus. Gaudard[22] expresses the view that diabetes in a mild form may set in at the beginning of pregnancy and disappear after labor, while a more severe form persists after delivery, and a still more serious variety rapidly terminates fatally. The condition, he finds, is aggravated by lactation. H. D. Fry[23] states that when a predisposition to diabetes exists pregnancy is likely to act as an exciting cause; when the disease is already present its type becomes aggravated. If a fatal termination does not take place before labor sets in, a change for the better may be expected at this time.

In a discussion of the principal complications of diabetes, Grellety[24] states that sexual indifference is habitual in consequence of the associated constitutional debility, and that impotence and sterility are a natural result. M. Loeb[25] relates that of 9 cases of diabetes mellitus in women under his observation 5 were complicated by disease of the uterus, namely, retroflexion, chronic metritis, fibroid, prolapse (2). M. Hofmeier[26] states that atrophy of the internal genitalia takes place in the sequence of diabetes mellitus in women.

In a discussion of diabetes in its relations with uterine activity, menstruation, and pregnancy Lecorché[27] calls attention to the fact

[21] Trans. of the Obstet. Soc. of London, 1882, xxiv, 256.

[22] Thèse de Paris, 1889; abstract in Centralbl. f. Gynäk., 1890, xiv, 24, 434.

[23] Trans. Amer. Gynec. Soc., 1891, xvi, 350.

[24] Lyon médical, 1880, xii, ann., t. xxxiii, 521.

[25] Berl. klin. Woch., October 10, 1881, 601.

[26] Ibid., October 15, 1883, 42, 641.

[27] Du diabète sucré chez la femme, Paris, 1886, 162 et seq.; extract in Annales de gynécologie, 1885, xxiv, 257.

that diabetes in women is observed especially before puberty and after the menopause, at the same time pointing out that the relative rarity of the disease appears counterbalanced by its gravity during this period. Both diabetes and the menopause are most common in women at about forty-four or forty-five years of age. On the other hand, diabetes is charged with causing more or less profound alterations in the function and structure of the uterus and its adnexa, so that frequently menstruation is irregular, painful, or absent, and conception is unlikely to take place. When pregnancy occurs the nutrition of the fetus is affected in such a manner that premature labor often takes place.

H. Senator[28] notes that menstruation becomes deranged in diabetic women only when nutrition is greatly impaired and that pregnancy has been observed to occur, although often interrupted by abortion. J. Seegen[29] found amenorrhœa an uncommon symptom in diabetic women, and he has observed normal menstruation, even in severe cases, up to within a week before death.

According to C. von Noorden[30] there is no general rule as to the occurrence of menstruation in diabetic women, amenorrhœa being noted early in some, while in others menstruation recurs at regular or irregular intervals until an advanced stage of the disease. He states that conception occurs frequently, even at a late period in the disease, and that pregnancy and the puerperium have no unfavorable effect on the diabetes, as illustrated by 5 diabetic women under his care who became pregnant. He admits, however, that in severe cases sexual desire is diminished and sterility likely to be present, and he advises against the marriage of young women having diabetes, as the disease is serious under such circumstances.

W. Lesse[31] believes that the prognosis is better in the case of a woman diabetic before pregnancy than in that of a woman who becomes diabetic during pregnancy. He considers the induction of premature labor advisable only in the presence of hydramnios or contracted pelvis. F. Schauta[32] considers diabetes a serious complication of pregnancy and especially during parturition. Accordingly, he would sanction the induction of abortion in the early months, as the fœtus is likely to die anyway; and also in the later months if the amount of sugar in the urine is large. G. E. Herman[33] favors the early termination of pregnancy as offering the greatest probability of benefit to the mother, especially if hydramnios be present. According to J. W. Williams[34] gestation in a diabetic woman sometimes exerts a deleterious influence on the course of the disease, and if the patient's condition should become alarming he advises the induction of premature labor.

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[28] Handbuch der speciellen Pathologie und Therapie, 1876, B. xiii, 2 H., 164.
[29] Der Diabetes Mellitus, 3 Aufl., Berlin, 1893.
[30] Die Zuckerkrankheit und ihre Behandlung, Berlin, 1898.
[31] Monat. f. Geburtsh. und Gynäk., 1902, xv, 680.
[32] Ibid., xvi, 470.
[33] Edinburgh Med. Jour., 1902, n. s. xi, 119.
[34] Obstetrics, 1903, 344.

P. Rudaux[35] states that diabetes retards the progress of pregnancy, equally whether it has been present before or has supervened upon conception. The diabetic woman seldom reaches full term; premature birth is more common than abortion before the child is viable. The amount of sugar increases about the sixth month and during the last days of the ninth month, and there is a definite relation between the amount of sugar and the death of the child. The fœtus may die in the uterus and be expelled subsequently; or, when it approaches term, it may die during labor or soon after birth. Death occurs during or after parturition in 41 per cent. of the cases. The most common complications are hydramnios and hydrocephalus; besides, the size of the child may be excessive. The effect of diabetes upon labor is unimportant, although uterine contractions are less energetic and, therefore, the period of parturition is prolonged. Diabetic patients are especially prone to septic infections, their tissues furnishing a favorable medium for the growth of pathogenic microörganisms. Lactation is well maintained and is accompanied with a diminution of the ordinary symptoms of thirst and polyuria. Part of the glucose is converted by the mammary glands into lactose, and a large proportion of the sugar found in the urine is lactose. On the other hand, pregnancy aggravates diabetes considerably, although the aggravation does not appear during the first four or five months. Toward the end of the fifth or during the sixth month the symptoms reassert themselves with increased intensity; the patient complains of thirst, and polyuria; glycosuria and emaciation become marked; or such complications as coma, loss of vision, paralysis, or syncope develop. The symptoms may have been so slight as to escape detection until aggravated by the pregnancy, or in cases that have been recognized prior to the occurrence of pregnancy the symptoms may have been dormant until the sixth month; but in either event the disease then makes rapid progress. In the latter days of pregnancy, should the patient go to term, the severity of the disease is greatly intensified; but after delivery some alleviation may be expected. Slight symptoms appearing toward the end of pregnancy and held in check by suitable treatment after delivery will be increased by a subsequent pregnancy. The average duration of diabetes is three or four years, but when complicated by pregnancy this is reduced to twenty months. The termination is usually due to coma, cachexia, pulmonary tuberculosis, or exhaustion, and it is often hastened by parturition. For an exhausted woman labor is a serious trauma, and sudden death or death within a few days after delivery will occur as a result of the shock. The effect of lactation can be looked upon only as harmful, as it prolongs the puerperal period and lowers the resistance of the maternal organism. When pregnancy occurs in a diabetic woman the prognosis is always serious for both the mother and the child.

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[35] La Clinique, October 19, 1906; Brit. Med. Jour., January 5, 1907; Epitome of Current Medical Literature, p. 2, No. 7

I have observed a case of diabetes in which the glycosuria disappeared with the inception of pregnancy and reappeared only after the delivery at about term of a macerated fœtus. I have been unable to find in the literature the record of a parallel case.

M. G., a married colored women, aged thirty-seven years, was referred to my dispensary service at the Polyclinic Hospital, on February 18, 1905, on account of vulvar pruritus and a suspicion of diabetes. Her mother had died at forty-nine years of age of some disease of the kidney and gangrene of the leg. Her father, two sisters, and one brother were living, in good health. She had borne one child, sixteen years previously, but it had lived only nine months. She had herself had measles in childhood. The woman related that she had been sick for three years, during which period she had been passing large amounts of urine at frequent intervals. Micturition was attended with burning pain and there was a purulent vaginal discharge, with marked swelling of the vulva.

For a year the lower extremities had been swollen; for six months she had had severe backache, and she had lost fifty pounds in weight in two months. The woman admitted that she had a fondness for sweets, on several occasions indulging herself to the extent of a pound and a half of candy in twenty-four hours.

The patient had at various times lost different finger-nails. She complained of want of appetite and nervousness. She drank water excessively. Menstruation was regular, attended with moderate pain for the first twenty-four hours, and lasted for three days. There was no history of mental or physical shock.

The woman weighed 206 pounds as compared with 220 rather more than a year previously. The action of the heart was rhythmic. The sounds were clear and the second was accentuated in the aortic area. The knee-jerks were preserved. The urine was found to be free from albumin, but sugar was present in a proportion of 4.67 per cent. From four to five pints of urine were passed in the twenty-four hours. Under treatment, in part dietetic, in part medicinal—salol, sodium phosphate—the patient began to feel better and to gain in weight. The pain on micturition diminished, the vaginal discharge and the itching grew less. Menstruation occurred on February 28, 1905, and a trace of blood from the uterus appeared about April 1. At the latter date morning nausea was noted and anorexia was marked. There was some soreness of the breasts, both of which contained secretion. The glands themselves had not increased in size, but the areolar tubercles were enlarged. Pregnancy was suspected, although this condition had not been present for sixteen years.

Urine examined on March 4 contained only 1.17 per cent. of sugar and another specimen examined on March 18 contained 1.6 per cent., while no sugar at all was found in specimens examined March 28, April 1, 8, and 29, and May 2. On November 15, 1905, the patient complained of tenderness and dull pain in the lower portion of the abdomen. The facial expression was dull and sleep was poor. Thirst was almost unquenchable. Urine was passed in large amounts. The specific gravity was 1005 and there was no response to tests for albumin and sugar. Labor set in definitely on the following morning, but it progressed so slowly that the membranes were ruptured and artificial dilatation of the uterine mouth was undertaken. The breech presented and expulsive efforts were feeble. By manipulation a foot was brought down and then the other foot, and finally the trunk was extracted. The head appeared to be impacted at the brim of the pelvis and the fœtus had to be turned several times before descent could be effected. The umbilical cord had broken at the fœtal extremity and the fœtus was dead and macerated. The placenta and membranes were delivered manually. Labor had occupied more than thirty-six hours and was followed by profuse uterine hemorrhage. The patient became comatose, with stertorous breathing, but she eventually reacted and recovered. On November 20 the urine contained a trace of albumin, but no sugar. On the 22d also no sugar was present, but a small amount of albumin was again found, together with red and colorless blood corpuscles, amorphous and triple phosphates, squamous epithelial cells, and free granules. On December 1 symptoms of thrombophlebitis in the left thigh appeared. The urine still contained a trace of albumin, but no sugar. On the 8th sugar was again not demonstrable in the urine, but a trace of albumin was present, with one hyaline cast and a few finely granular casts, as well as leukocytes, crystals of uric acid and of calcium oxalate, squamous epithelial cells, and free granules. The subsequent history of the patient may be summed up briefly in the statement that the albumin disappeared from the urine, while in April, 1906, sugar was again found and the symptoms of diabetes previously present once more made their appearance The patient's weight, however, increased to 226½ pounds.

The patient was skilfully looked after during labor and the puerperium by Dr. Annie L. Conner, to whom I am indebted for notes covering that period of the case.

I append herewith abstracts of the cases that I have collected from the literature, giving separately those in which pregnancy was complicated by diabetes and those in which pregnancy took place in diabetic women. I have endeavored further to separate the cases in which diabetes recurred in successive pregnancies and those in which diabetes appeared during pregnancy after having been present temporarily at an earlier period.

I. CASES OF PREGNANCY COMPLICATED BY DIABETES.

J. M. Duncan[36] reports the case of a woman, aged thirty years, in the fifth month of her third pregnancy. She was married at twenty-six years, and her first child was born, decomposed, at term. Subsequently she presented a succession of boils, but her urine was not examined. Her second child was born, also decomposed and at term, fifteen months later, and again the urine was not examined. The woman became pregnant a third time six months later and now the urine was found "diabetic." Alarming symptoms developed in the seventh month and premature labor was induced. Improvement followed for a time, but later a fatal termination ensued.

It can be only a matter for speculation as to whether diabetes was present in the first pregnancy in this case, and whether it continued and was present also in the second pregnancy.

M. Loeb[37] reports the case of a woman, aged twenty-two years, who presented symptoms of diabetes in the second month of pregnancy. Abortion took place in the sixth month and death followed soon afterward.

In a paper on "Puerperal Diabetes," J. M. Duncan[38] reports 2 additional cases of his own and also 11 others from various sources, besides those of Bennewitz and Lecorché. In one of Duncan's cases, in a woman aged thirty years, there was a suspicion of temporary diabetes at the end of a former pregnancy. In the fifth pregnancy diabetes was discovered in the eighth month and the fœtus died before labor set in, at the beginning of the ninth month, with collapse. There was an excess of liquor amnii. Death took place on the third day after delivery.

Duncan's second case occurred in a woman, aged thirty-five years, in whom diabetes was discovered at the time of quickening in her eleventh pregnancy. A large child was born dead, and the symptoms of diabetes disappeared. Later, however, there was a relapse, and death took place in coma eight months after delivery.

The case of W. L. Reid (Duncan) occurred in a woman, aged thirty-two years, whose first pregnancy terminated in the delivery of a macerated fœtus. Diabetes was discovered early in the second pregnancy. The fœtus died in the eighth month and premature labor occurred a few days later. The liquor amnii was excessive. The diabetes persisted.

A case of Williams (cited by Duncan) occurred in a woman, aged thirty-six years, who developed diabetes early in her seventh pregnancy. The fœtus died during pregnancy and was expelled in the eighth month. The mother died suddenly four months later.

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[36] Edinburgh Med. Jour., 1873, xviii, ii, 696.

[37] Berl. klin. Woch., October 10, 1881, 601.

[38] Trans. of the Obstet. Soc. of London, 1882, xxiv, 256.

Another case of Williams (cited by Duncan) occurred in a woman, aged thirty-three years, who first became pregnant at the age of twenty years, and exhibited thirst and emaciation, but became strong again after delivery. Her second pregnancy began when she was twenty-two years old, and was attended with the same symptoms. Her third pregnancy, which occurred when she was twenty-three years old, was attended with slight loss of flesh. There was a fourth pregnancy at twenty-five years, a fifth at twenty-eight years, and a sixth at thirty years. In the last there were again thirst and emaciation and incontinence of urine. The seventh pregnancy took place at thirty-three years, and its course was marked by wasting, thirst, and incontinence of urine. Glycosuria was discovered a short while after delivery, disappearing in the course of three months.

It is possible that in this case diabetes was present in the first second, third, and sixth pregnancies, but in the absence of any statement with reference to the urine the necessary certainty for the formation of an opinion is wanting. Although the discovery of sugar in the urine soon after the last labor renders it likely that glycosuria was present during the seventh pregnancy, the omission of a specific report on this point leaves room for reasonable doubt.

Husband's case (Duncan) occurred in a woman, aged twenty-seven years, in whom diabetes was discovered in the third pergnancy. The child was feeble at birth and died a few hours later. The liquor amnii contained sugar. The mother died eight months after delivery.

In Winckel's case (Duncan) diabetes was observed in the second pregnancy and the child was born alive.

In Davidson's case (Duncan) a woman, aged thirty-eight years, was found to be diabetic in the middle of her fourth pregnancy. Labor set in prematurely and the child lived for thirteen hours. The symptoms of diabetes disappeared, but returned, with a fatal termination, four months later.

Frerich's case (Duncan) occurred in a woman, aged thirty-six years, in whom diabetes was discovered in the eighth month of her tenth pregnancy. The lying-in was normal. Death took place fifteen months later from tuberculosis and gangrene of the lungs. On postmortem examination a tumor of the medulla oblongata was found.

L. Kleinwächter[39] reports the case of a woman, aged twenty-two years, who aborted a few weeks after marriage and during her second pregnancy presented symptoms of severe diabetes. In the thirty-second or thirty-third week amniotic fluid in excessive amount escaped and the patient went into collapse. Forceps were applied and an immature fœtus was delivered. The child lived for but a short time and the mother died eighteen hours after labor. Kleinwächter believes the disease must have commenced in the twentieth year.

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[39] Wien. med. Presse, 1904, xlv, 51, 2448.

M. J. Durieux[40] reports the case of a woman, aged thirty-seven years, whose first pregnancy, attended with albuminuria and eclampsia, resulted at term in the birth of a dead infant. Six weeks after labor all trace of albumin had disappeared from the urine. The second pregnancy was interrupted in the second month by abortion without appreciable cause. Symptoms of diabetes made their appearance in the seventh month of the third pregnancy. A healthy child was born at term, and the urine was free from albumin and sugar after disappearance of the lochia.

B. Naunyn[41] refers to the case of a woman, aged thirty-nine years, who presented symptoms of diabetes in her ninth pregnancy and four years later still had pruritus and glycosuria.

H. D. Fry[42] reports the case of a woman, aged thirty-one years, who exhibited symptoms of diabetes in the fifth month of her second pregnancy. The fœtus died in the seventh month and premature labor followed. The mother died on the fifth day.

Born[43] reports the case of a woman, aged twenty-four years, who in the seventh month of pregnancy died in diabetic coma. The uterus contained a macerated fetus.

E. L. Partridge[44] reports the case of a woman, aged twenty years, who developed diabetes in the fourth month of her first pregnancy, the disease subsiding in the seventh month, and labor taking place at term, with the delivery of a healthy child. The urine was free from sugar one month afterward.

E. Rossa[45] reports the case of a primipara, aged twenty-eight years, with a rachitic contracted pelvis, whose urine in the sixth month of pregnancy yielded a distinct reaction for sugar, which was found to be due to dextrose. The fœtus was living and exhibited hydramnios. The patient declined Cesarean section, so that premature labor was induced. The membranes were ruptured and three liters of amniotic fluid was evacuated. Delivery ensued sixteen hours later, but the fœtus lived for only thirty-two hours. Grape sugar was found in the amniotic fluid, but not in the fœtus' urine. The sugar disappeared from the mother's urine six days after labor. The case is considered one of mild diabetes superinduced by pregnancy.

G. E. Herman[46] reports the case of a woman, aged thirty-two years, who had borne seven children. All of the labors were difficult and the last three children were stillborn. In the third month of her eighth pregnancy the woman was weak, with an irritating vaginal discharge, burning micturition, itching, and boils. For three months there had been thirst, as there had been also in previous pregnancies, and for one month loss of flesh. The woman had been told in the fourth month of pregnancy that she had diabetes. When seen in the seventh month she had bearing-down pains and vomiting, and in a few days she became moribund. The urine contained albumin and sugar. Fluctuation was present over the uterus. The fœtal heart sounds could not be heard, nor fœtal movements felt. The membranes were ruptured and a large amount of amniotic fluid escaped. Natural delivery of a decomposed fœtus took place. The amniotic fluid was excessive and contained albumin and sugar. The woman died ten days later.

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[40] Bull. de la Société d'Obstét. de Paris, 1905, viii, 141.

[41] Der Diabetes Mellitus, Vienna, 1906, 234.

[42] Trans. Amer. Gynec. Soc., 1891, xvi, 350.

[43] Correspondenzblatt f. Schweizer Aerzte, 1892, xxii, 347.

[44] Med. Rec., July 27, 1895, 109.

[45] Centralbl. f. Gynäk., June 20, 1896, 25, 656.

[46] Edinburgh Med. Jour., 1902, n. s. xi, 119.

In the absence of a knowledge of the state of the urine no opinion is possible as to whether diabetes was present in any of the pregnancies but the last.

A. Stengel[47] reports the case of a woman, aged thirty-three years, in whom symptoms of diabetes were observed in the fourth or fifth month of her second pregnancy. A healthy child was born at term, and the glycosuria disappeared one month after labor.

II. CASES OF DIABETES IN SUCCESSIVE PREGNANCIES.

Bennewitz[48] reports the case of a woman who in her fourth pregnancy suffered from insatiable thirst and polyuria, which ceased after delivery. The urine was not examined. During her fifth pregnancy, at the age of twenty-two years, she presented the same symptoms and the urine examined at this time contained "two ounces of saccharine matter to the pound." Labor set in prematurely and the woman gave birth to a twelve-pound infant, which died in the process of delivery. The woman was well again in a short while and the urine free from sugar. Six months later she became pregnant for the sixth time, with the same symptoms as she had previously exhibited, but these all disappeared after delivery. It is possible that diabetes was present during the fourth pregnancy as well as during the fifth and the sixth.

A case of Williams (cited by Duncan) occurred in a woman, aged twenty-five years, in whom diabetes was discovered the day following the birth of her first child at term. The sugar gradually disappeared from the urine, but it reappeared in the succeeding pregnancy, which terminated in the birth of a healthy child, with an excess of liquor amnii, in which, however, no sugar was found.

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[47] Univ. of Penna. Med. Bull., October, 1903.

[48] Osann's 12. Jahresbericht des poliklinsichen Instituts zu Berlin, p. 23; Edinburgh Med. Jour., 1828, xxx, 217.

Sugar was present again in the third pregnancy, at the termination of which the mother was safely delivered of a small child. Death took place, however, one month later, in coma.

This case belongs also in the next category, being one in which the diabetes recurred during pregnancy after having previously been present temporarily.

F. A. Packard[49] reports the case of a woman, aged forty-one years, in whom symptoms of diabetes were present during each of four pregnancies, disappearing, however, in the intervals, but persisting after the last. The gravity of the symptoms increased progressively with each succeeding pregnancy and all terminated in miscarriage.

M. A. Tate[50] reports the case of a woman, aged thirty-five years, in whom symptoms of diabetes were observed in the third month of her second pregnancy and miscarriage took place in the fourth month. Sugar had disappeared from the urine one month afterward. Similar symptoms appeared in the third month of the third pregnancy, the course of which, however, was not interrupted. Labor terminated normally and the sugar disappeared from the urine.

III. CASES IN WHICH DIABETES RECURRED DURING PREGNANCY AFTER HAVING PREVIOUSLY BEEN PRESENT TEMPORARILY.

M. Graefe[51] reports the case of a woman, aged thirty-seven years, who several years following the birth of her sixth child presented symptoms of diabetes, which, however, disappeared under treatment. Fifteen months later she became pregnant for the seventh time and the symptoms of diabetes returned. In the seventh month fœtal movements ceased and a dead fœtus was delivered. The mother improved for a time, but subsequently grew worse.

W. Liepmann[52] reports the case of a sextipara, aged thirty years, with a previous history of diabetes mellitus, but whose labors had always taken place normally. Polyuria, glycosuria, and albuminuria, with hydramnios, were discovered about the fifth month of pregnancy. Two months later the membranes were ruptured and 11 or 12 liters of fluid evacuated. The fœtus was extracted with some difficulty and copious postpartum hemorrhage ensued. The sugar disappeared from the urine for a short time, but soon reappeared, and death took place nine days after delivery. In addition to degeneration of the kidneys, arteriosclerosis, and gastric ulcer, dissecting metritis was present.

J. Fels[53] reports the case of a woman, aged thirty-four years, who some time after her third pregnancy presented symptoms of diabetes. These cleared up under treatment, but reappeared later, in a fourth pregnancy, again disappearing under treatment. The woman was delivered of a normal living child at term, but died three years later, in coma.

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[49] Univ. Med. Mag., 1888–89, i, 229.

[50] Amer. Jour. of Obstet., January, 1906, liii, 65.

[51] Sammlung zwangloser Abhandlungen aus dem Gebiete der Frauenkrankheiten und Geburtshilfe. 1898.

[52] Archiv f. Gynäk., 1903, lxx, 426.

[53] Wien. med. Presse, 1905, 13, 627.

IV. CASES OF PREGNANCY IN DIABETIC WOMEN.

Lecorché[54] reports the case of a woman, aged twenty-eight years, who was found to be diabetic following the birth of a child six years previously. The infant also became diabetic. Two years subsequently the woman became pregnant again and gestation went on to favorable termination.

Two cases of Newman are cited (Duncan). Both occurred in women, aged thirty-six years. In one diabetes developed during or perhaps before the first pregnancy, which terminated in normal labor, and it persisted throughout two succeeding pregnancies. The second pregnancy and parturition also were normal. In the third pregnancy the child was born dead at six or seven months and the mother died three days later. In the second case diabetes was observed in two pregnancies. The children were born alive, but the mother died in coma two years after the birth of the last child.

Duncan cites a case of Seegen in which three pregnancies occurred during the course of diabetes. All ended in miscarriage in the fourth or fifth month, with death of the mother after the last.

F. Minot[55] reports the case of a woman, aged twenty years, who in the fifth month of pregnancy was seized with vomiting after a hearty meal, gradually became unconscious and died two days later. When first seen, six months previously, she presented the usual symptoms of diabetes, passing, in twenty-four hours, seven pints of urine containing large amounts of sugar.

H. Ludwig[56] reports the case of a woman, aged forty-two years, who in her ninth pregnancy presented symptoms of diabetes of two years' standing. In the eighth month the abdomen became greatly distended and acetone and diacetic acid appeared in the urine in addition to sugar. The membranes were ruptured and an excessive amount of amniotic fluid evacuated, followed by a macerated fœtus. The mother's condition steadily improved. The amniotic fluid contained dextrose (0.30 per cent.).

In the course of a discussion before the Obstetrical Society of London, Griffith[57] referred to a case of diabetes of some years' standing, in a woman who became pregnant and passed through pregnancy and labor without any sign of danger.

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[54] Archives générales de médecine, 1882, i. 385.

[55] Boston Med. and Surg. Jour., January 19, 1888, 69.

[56] Centralb. f. Gynäk., March 16, 1895, 11, 281.

[57] Lancet, June 23, 1906, 1761.

An analysis of the foregoing cases shows that in the vast majority, namely 27, diabetes occurred as a complication of pregnancy, while in only 8 did pregnancy occur in diabetic women. Among the former diabetes recurred in four successive pregnancies in 1 case, and in two successive pregnancies in 3 cases. In 3 cases diabetes recurred during pregnancy after having previously been present temporarily. Two diabetic women became pregnant thrice and 1 other pregnant twice.

The association of pregnancy and diabetes was noted at almost all ages between twenty and forty-two years, without especial prevalence at any one period. It occurred most commonly in the first, second, and third pregnancies, although it has been observed also in later pregnancies, up to the eleventh. The existence of the diabetes was recognized in the majority of cases in the first half of pregnancy, although in these, as in the others, it may naturally have existed prior to its detection.

Death of the fœtus resulted in the predominance of the cases, namely, in 28 as against 11, either during the continuance of pregnancy or as a result of miscarriage or premature labor, spontaneous or induced. In a considerable number—8—hydramnois existed, the amniotic fluid containing sugar in 4 instances. One infant became diabetic.

Of the mothers 19 died at varying periods following delivery, and 4 of these after temporary improvement. In 3 the diabetes persisted, in 1 it grew worse after temporary improvement. In 7 cases diabetes was present in successive pregnancies. Improvement or recovery took place in 10 cases. My own case is unique in the disappearance of the glycosuria with the advent of pregnancy and its reappearance a short time after delivery.

Summary. There is evidence to show that the power of assimilating carbohydrates is diminished in conjunction with pregnancy, and this deficiency may be manifested by the appearance of sugar in the urine.

Transitory glycosuria toward the end of pregnancy or during the puerperium may be considered physiological and related to the activity of the mammary glands in the secretion of milk; therefore, a resorption phenomenon.

Diabetes is an uncommon complication of pregnancy, partly because the disease is, in general, less common in women than in men, but principally because diabetes occurs, as a rule, at a later period of life than pregnancy usually takes place. Occasionally diabetes has been noted in two or more successive pregnancies.

Pregnancy occurs even more rarely in diabetic women, partly because, in addition to the reasons already given, of the depraved nutrition of the diabetic patient and of the disordered function and structure of the internal generative organs.

The complication of the one condition with the other usually increases the gravity of both. Often the fœtus dies *in utero*, and miscarriage or premature labor takes place, or the child may die during birth or shortly afterward. The child also may be diabetic. The liquor amnii has been found increased, and in some instances also to contain sugar. The mother often dies of the disease, sometimes during pregnancy or in childbirth or shortly afterward.

Accordingly, a diabetic woman should not marry; or if married she should not become pregnant. In the event of a diabetic woman becoming pregnant or of a pregnant woman becoming diabetic, pregnancy need not be interrupted artificially unless some special indication arises. If the woman is safely delivered she should not nurse her child.

HYPERTHYROIDISM.

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A typical case of Graves' disease, with the cardinal and subsidiary symptoms all characteristically grouped, is diagnosticated without difficulty. In the pure type of the disease, the five cardinal symptoms occupy the foreground of the clinical picture with the various secondary functional manifestations present, but less constant and intense. The diagnosis becomes somewhat difficult if the secondary symptoms are predominant, with but one or two cardinal indications present. Most recent writers on the subject refer, with more or less emphasis, to this class of cases, and there is an apparent disposition to distinguish a mild and temporary from the essential or chronic form of Graves' disease. The attempt to draw a sharp line of distinction has little vindication from practical experience, since we are constantly observing a gradual transition and eventual termination of the mild in the severe and fully-developed disease. In the clinical valuation of the different grades of thyroid toxemia, or Graves' disease, the matter stands much in the same light as the inter-relationship of the glycosurias and diabetes. It is only in perspective that we are able to estimate whether a functional perversion is the expression of some essential malady, or merely a mild and transient reaction to irritation. The necessity for the proper recognition of these atypical forms of Graves' disease is great, in consideration of the fact that the disease, so far as we can now tell, is one and the same, whether we call it complete or incomplete—difference being a matter of degree only. Various names have been applied to these mild types of the disease. They have been designated "aberrant types," "pseudo-Graves'," "formes fruste," "larval types." The term "hyperthyroidism" has often been employed to characterize states of thyroid toxemia which are apparently less than Graves' disease. It is with these subtoxic states that the present paper is concerned.

The frequency of occurrence of morbid overactivity of the thyroid gland is probably much greater than is generally believed; for, with the present better appreciation of the nature of thyroid intoxication, many cases are being thus interpreted that in other times were probably passed over as instances of neurasthenia, hysteria, functional heart disease, anemia, etc. My own experience has brought to my attention with strong emphasis the importance of thyroid toxemia as a complicating element in clinical work.

The question of the pathogenesis of Graves' disease seems at last settled in favor of the theory of Möbius, which postulates an overactivity or perverted activity of the thyroid gland as the essential feature. The vasomotor, nervous, and cardiac symptoms are toxic phenomena, dependent on the presence in the blood of excessive amounts of thyroid secretion or of thyroid secretion of abnormal quality. The primary cause of the thyroid disturbance is still left unexplained, although there seems little doubt that it is a toxic reaction, dependent on some morbid material as yet unidentified. The whole structure of the surgical treatment of Graves' disease rests upon the theory of hyperthyroidism, and if it be not true, then the hundreds of thyroidectomies which are now matters of record remain without justification. The striking influence over the disease, which follows a well-executed partial thyroidectomy, furnishes evidence in favor of the theory of hyperthyroidism, which is so direct as to be apparently irrefutable. The rapidly accumulating surgical records in Graves' disease furnish a clinical document that has few parallels in the history of modern medicine.

The results of thyroid feeding supply evidence which is hardly less conclusive. The administration of thyroid gland substance, or thyroid extract, is capable, if given in sufficient amount, of inducing a toxic state, which in almost every essential is similar to Graves' disease. An artificial state of hyperthyroidism is thereby produced, which duplicates almost in full the morbid syndrome. Even the characteristic exophthalmic symptoms have been observed after thyroid feeding by Auld, Béclere, and others, and Edmunds was able to induce proptosis, widening of the palpebral fissure, and dilatation of the pupils, in six monkeys, by this means, even after excision of a portion of the cervical sympathetic.

The following clinical instance of artificial thyroid toxemia constitutes a striking example of the effect upon the human: My patient is a young man, aged twenty-two years. He was referred to me for general examination by Dr. Thomas A. Woodruff, of Chicago, who had found low grade degenerative changes in the choroid, such as are produced by the chronic intoxications. Omitting all details of the case, which have no bearing on the purpose of the present citation, it will suffice to state that the patient has a small, simple goitre, which has existed without symptoms since childhood. A few weeks before examination he experienced for the first time a sense of swelling and constriction of the throat, excessive sweating, and a good deal of restlessness and general nervousness. Examination revealed a well-developed thyroid toxemia, marked by restlessness, hyperidrosis, exaggerated reflexes, tachycardia (pulse 100 to 126), excessive pulsation of the carotids and femorals, fine tremor of the hands, tongue, and closed eyelids. There was no exophthalmos. It appeared that four months before the date of consultation, he had been induced to take thyroid extract to remove his goitre, and, not observing any effect upon the tumor, had gradually and steadily increased the dosage, believing the stuff harmless. From 15 grains daily—the initial dose—he had attained an average of 75 grains a day, and affirmed that he had taken 200 5-grain tablets (1000 grains) during the preceding week. The maximum daily quantity taken on any one day was 150 grains. Strange to say he had gained perceptibly in weight during the time he took the preparation. Discontinuance of the drug, under my directions, resulted in a prompt subsidence and final disappearance of the toxic symptoms.

The exact part that the thyroid plays in systemic metabolism is, unfortunately, still largely a matter of conjecture. Certain empirical physiological facts have been accumulated by clinical observation, which apparently have a bearing on the incidence of thyroid functional perversions. That the gland exerts an influence on the development of the organs of generation of both sexes and is affected by the vicissitudes of the sexual life of the individual, is demonstrated by many observations. The asexuality and infantilism of cretins and the different cretinoid states is well known. The intimacy of relationship between the thyroid and the generative organs is much greater in the female than in the male, probably owing to the recurring functional demands of menstruation, pregnancy, and lactation, and we consequently encounter thyroid disturbance with much greater frequency in women than in men. In many women the thyroid enlarges at the first menstruation, and at each subsequent period, producing at times an appreciable enlargement. It is stated that in girls the commencement of the sexual life is accompanied by an enlargement of the thyroid, and the gland has been observed to enlarge during rut in the cat, rat, sheep, and deer. Suppression of the menses may produce an enlargement of the thyroid, which subsides on their re-establishment. It is stated by Richardson that in Southern Italy it has long been the custom for the parent to measure carefully the circumference of the daughter's neck before and after marriage—an increase in size being considered an evidence of pregnancy. Thus the thyroid gland is subject to congestion and functional hypertrophy under the normal conditions of puberty, menstruation, marriage, and pregnancy. Although physiological in their inception, these thyroidal disturbances may at any time, under persistent or excessive stimulation, pass the borderline and become pathological, causing symptoms of the Graves type.

The following instances from my case records exemplify this transformation: A little girl, aged eleven years, was brought for examination by her mother, who was concerned over an enlargement of the child's neck which had appeared in December last. Menstruation had occurred first in February, two months after the appearance of the goitre, and again in March and April. It had been observed that the tumor was larger at the menstrual periods than at other times. On examination it was found that in addition to a small bilateral goitre, there was a well-marked fine tremor of the hands and slight irregular twitching of the extremities, sweatings, flushings, morbid blushing, general nervousness, and mental depression. The pulse was persistently above 100; temperature 99.5°. It is interesting to note the fact that in this case the thyroid enlargement antedated puberty. Broca and Richardson suggest that as the thyroid apparently presides over the development of the generative organs, its failure to secrete being marked by infantilism and sexual involution, it might be serviceable to administer thyroid gland substances in cases of delayed puberty.

Another case of somewhat different character and greater interest is that of a young lady of Syrian birth, now nineteen years of age. I might note the fact in passing that, after a considerable experience during three years' work among the Syrian colony in Chicago, more than one-half the women of this nationality I have seen have enlarged thyroids. They are otherwise normal and well developed. The girl under reference has had a small-sized simple goitre since early childhood. Three months previous to consultation she began to experience backache, dizziness, palpitations, dyspnœa, hot flushes, sweatings, insomnia, and gradually lost in body weight. Menstruation since fourteen; regular, but scant. Examination revealed marked throbbing of the neck vessels and abdominal pulsation, exaggerated reflexes, dermographia, moist skin, fine tremor of the hands, lid, and tongue tremor. Blood examination showed slight anemia; urine normal. During ten weeks' observation, the pulse was never observed below 100. Average systolic blood pressure, 100 mm. (Stanton). At the end of this period of observation the patient married. Within a week after marriage all symptoms had abated, and she rapidly put on flesh, regaining all her lost weight (eight pounds). She remained entirely free from the former troublesome symptoms for two months when she became pregnant. This has had the effect of causing a reappearance of the thyroid toxemia. The rapid disappearance of toxic symptoms after marriage, it seemed to me, very evidently indicated that the nervous stress and sexual stimulation of courtship were the exciting factors in causing hyperthyroidism. Re-appearance of the symptoms with the advent of pregnancy is not surprising, as we well know that during pregnancy there is a marked increase in thyroid activity. Freund observed augmentation in the size of the gland in 45 of 50 pregnant women, and Lange in 108 of 133 cases examined. In myxedematous women a marked improvement during pregnancy has been observed—the increased thyroid secretion being utilized by the mother to the probable detriment of the child. The functional stimulation of the thyroid incident to pregnancy usually extends throughout lactation, at the end of which period this physiological hypersecretion subsides without detriment to the gland. Occasionally it may serve as the starting point of a genuine Graves' disease, or, on the other hand, excessive thyroid involution may result, producing myxedema. Rapidly succeeding pregnancies with their attendant lactations, are particularly hazardous to the welfare of the gland.

The following case is cited from personal observation: A woman, now thirty-six years of age, has been married eight years, and during the first six years of her married life had five successful pregnancies, one a twin birth. She has always done her own housework and nursed her children. She observed a swelling of the neck four years ago. She complained of early and excessive fatigue on exertion, poststernal heaviness, frequent palpitation, dyspnœa on effort, evening malleolar œdema, sweating, backache, anorexia, excessive urine, and frequent micturition. Menstruation regular. Physical examination showed a pulse of 118; reacting excessively to effort and change of position. Systolic blood pressure, 135 mm.; diastolic, 105 mm. (Stanton). Exaggerated knee-jerks; moist skin; fine rhythmical tremor. There is distinct cardiac enlargement; no exophthalmos. A small uniform bilateral goitre exists.

One of the cases cited represents the type of thyroid toxemia arising in the presence of a simple goitre of long standing. It is far from uncommon to observe transient periods of tachycardia, tremor, sweating, and emotionalism in cases of ordinary goitre. Bloodgood[1] has recently called attention again to this association. Rarely in this group do we observe exophthalmos, the toxemia being manifested by nervousness, tremor, and tachycardia. These symptoms have been observed in simple goitre, thyroid cysts, adenoma, carcinoma, and tuberculosis of the gland. These toxic outbursts are usually comparatively mild, but are undoubtedly of the nature of Graves' disease. Bloodgood states that, as a rule, in cases of this character one will find cellular changes in the thyroid tissue identical with those of Graves' disease. Appreciation of the true significance of these toxic symptoms is of much practical importance to the surgeon. Cases of goitre of whatsoever form, which exhibit symptoms of this character, are to be operated upon with caution, owing to the greatly increased liability to the development of post-operative thyroidism in such subjects. Clinical observations of this character certainly furnish a very strong presumption in favor of the identity of hyperthyroidism and Graves' disease—the difference being mainly a question of dosage.

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[1] Surgery, Gynecology, and Obstetrics, August, 1905.

In addition to the foregoing types of hyperthyroidism, which are physiological in their inception, there are met with at least two other forms of thyroid hyperactivity, which are secondary to definite pathological states elsewhere in the economy. In many cases of valvular heart disease, particularly lesions of the mitral valve, whether primary or secondary to myocardial weakess, the thyroid may undergo enlargement from venous engorgement, and symptoms of hyperthyroidism may result. Much practical difficulty may arise in the more pronounced instances of this character in determining whether the thyroid enlargement and toxemia are the result of the valvular lesion, or its cause.

Another type of hyperthyroidism of great clinical interest is toxic hyperthyroidism. The simplest variety of this class of cases is the fulness of the neck, palpitations, tremors, sweatings, and depression, so frequently observed during the course of the anemias. Most cases of pronounced chlorosis are accompanied by some manifestation of this nature. In those of highly nervous temperament, especially women of hysterical tendencies, any form of systemic toxemia may awaken a morbid degree of activity in the thyroid, manifested by fulness of the neck and the characteristic vasomotor and nervous phenomena. Digestive toxemia, syphilis, diabetes, Bright's disease, may be so accompanied. I cite the following instance of its association with Bright's disease. There is not lacking an hereditary and personal predisposition to thyroid perversion in this patient. A sister died of Graves' disease and cardiac dilatation under my observation, and the patient gives a history of a year's ill-health at the age of eighteen, characterized by dyspnœa, palpitations, and obscure nervous manifestations. She remained in good health until four years ago, when albuminuria developed and chronic nephritis was diagnosticated. The case has been a high blood pressure one, with polyuria, dyspnœa of effort, but never any œdema or uremia. Four months ago a goitre appeared and has persisted, at the present time constituting a moderate-sized bilateral tumor. Rapid loss of weight followed—until the deficit amounts to 61 pounds (from 165 to 104 pounds). There was sweating, palpitation, pigmentation of the surface, tremor, morning headaches, and mental depression. No exophthalmos exists. The fundus oculi shows choroidal degenerative markings and vascular alterations. The heart is markedly enlarged, a faint systolic whiff is audible in the mitral area. The pulse varies from 110 to 130, very irregular in rhythm. Blood pressure: systolic, 165 mm.; diastolic, 100 mm. (Stanton). The urine shows a considerable albuminuria, and degenerative renal products.

Tuberculosis is frequently accompanied by symptoms which strongly suggest a thyroid source, and, in practical experience, it is not uncommon to meet with mild hyperthyroidism during the incipient stages of the disease. The two conditions resemble each other not a little, having in common slight pyrexia, sweating, flushing, increased reflexes, rapid pulse, and decline in body weight. It is not unlikely that many cases of hyperthyroidism in which no actual thyroid enlargement exists are wrongfully suspected of being incipient tuberculosis. The mistake is a natural one at a stage when sputum is not available for bacteriological diagnosis. A misjudgment in the opposite direction would be a much more prejudicial clinical error. How difficult the problem may be is illustrated by the following case: A young woman, aged twenty-two years, by occupation a clerk, consulted me several months ago. There is a good family and personal history; hygienic surroundings are average, although her occupation is confining. One year previously she had an acute infection of unknown character, marked by repeated chills and pyrexia, and it is from this attack that she dates her ill-health. Since then she has been weak and nervous, has troublesome hot flushes and hyperidrosis, so that she is constantly wiping her face and hands. There is troublesome palpitation, insomnia, headache, backache, dyspnœa, sternal pain and weight, and a slight hacking cough in the morning. Menses irregular. Steady decline in weight. There is an afternoon temperature varying from one-half to one degree above normal, increased by bodily activity and slightly greater during menstrual periods.

Examination shows a slightly enlarged heart and faint systolic bruit. There is a slight fulness of the neck; the pulse varies from 100 to 130. Average systolic blood pressure, 120 mm. (Stanton). There is well-marked fine muscular tremor of the hands and eye lids. Urine normal. Blood examination shows slight simple anemia. The sputum, which is scant, was examined several times with negative results. I would have been inclined to regard this case as the hyperthyroidism of menstrual irregularity in an hysterical woman had it not been for a small area in the upper part of the left lung, which left a doubtful impression on examination. Close observation of this focus covering a period of two months enabled me to watch its development into a typical area of tuberculous involvement.

It is not at all improbable that the thyroid secretion plays a part in the resistance offered by the organism against acute infectious diseases. We may infer as much from the frequency with which swelling of the gland is observed in acute infections of microbic origin. Statistics are not wanting in this connection.

Vincent[2] draws attention to painful swelling of the thyroid in 50 per cent. of his cases of acute articular rheumatism with fever.

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[2] Bulletin et Memoirs Soc. Méd. de Paris, 1906, xxiii, 598.

The swelling is most marked at the outset, and when the gland is not spontaneously painful it is tender to pressure. The swelling and tenderness disappear with improvement of the general symptoms. Vincent does not regard this as in any way peculiar to rheumatism, as it occurs in other infections as well. He has found the same thing in 11 of 17 cases of typhoid fever, in 1 of 4 cases of meningitis, in 7 of 15 cases of measles, in 9 of 19 cases of scarlet fever, in 1 of 9 cases of mumps, in both of 2 cases of erythema nodosum, and in 2 of 5 cases of acute malaria. These observations would certainly lead us to believe that the thyroid plays a part in the defence of the organism against acute microbic processes, and to infer that the gland is stimulated to increased physiological activity by toxins which its secretion is in some way destined to neutralize (Marin).

A possible sidelight on the connection between hyperthyroidism and Graves' disease is forthcoming from a consideration of the following statistics of the association of acute rheumatism and Graves' disease: Robinson[3] reports 127 cases of Graves' disease, 18.9 per cent. of which gave a definite previous history of acute rheumatism. West, in 1886, reported 38 cases of Graves' disease, 8 of which had a history of acute rheumatism. In 1890, MacKenzie collected 40 cases, 9 of which gave a history of quinsy and 5 acute rheumatism. In 1893, Thompson reported 8 cases of Graves' disease, 3 of which had had acute rheumatism. In view of these suggestive figures, taken in connection with the statistics of Vincent, it is incumbent on us to investigate the personal history of every case that comes under observation with especial reference to preceding acute infections. We are coming more and more to realize the deleterious late after-effects of acute microbic infections. The offence which is offered to the systemic tissues by the severe infectious toxemias is not one which is quickly condoned. We are much too apt to minimize the etiological importance of acute infections occurring in the antecedent history of patients who are suffering from obscure organic dystrophies and nutritive disorders. Many cases of organic disease of the liver, kidneys, vascular system and heart, as well as cases of diabetes and the essential anemias, can, with a fair degree of certainty, be traced back to an acute infection occurring, perhaps, many years previously. In consideration of the prominent part played by the thyroid in the resistance of the organism to acute disease, and the frequency of severe congestions of the gland, as shown by the observations of Vincent, we may, with reason, hypothesize some connection between acute microbic infections and Graves' disease. A severe toxic hyperthyroidism or a prolonged functional strain upon the organ, such as results from repeated pregnancies, anemia, etc., may beget a morbid irritability of the thyroid, which merely awaits the exciting factor of a sudden functional demand, resulting from shock, fright, or severe emotional disturbance, to establish the pathological type of thyroid overactivity known as Graves' disease. A careful, systematic inquiry into the previous history of every case of Graves' disease, with a view to tabulating the acute infections, neuropathical disturbances, anemias, menstrual disorders, etc., might yield suggestive data toward a better understanding of the etiology of this obscure disease.

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[3] Lancet, 1906, i, 1037.

PYELONEPHROSIS OF A SUPERNUMERARY KIDNEY.

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Mr. C., a medical student, aged twenty-three years, entered the Carney Hospital in September, 1906. Ever since he was three months old a swelling, the size of a hen's egg, had been observed in the left lower quadrant of his abdomen. From his third to his eighteenth year he had frequent attacks of "indigestion" following indiscretions in diet. The symptoms of these attacks were general abdominal pain, vomiting, and fever, and a tumor would appear in the left lower quadrant, last three or four days, and disappear when the bowels moved. During the last five years he has been free from attacks of this character. Four months before he came to Boston, after an illness of four days, he entered a hospital in a neighboring city. He complained then of headache, fever, malaise, constipation, and dull, bearing-down pains over the bladder. He had no vomiting or chills. A large globular mass was felt rising above the pelvis nearly to the level of the umbilicus. This tumor resembled a distended bladder, but when the latter was catheterized, the tumor merely sank back toward the sacrum. It was tender to pressure, especially to the left of the median line. There was some spasm of the rectus. There was a leukocytosis of 12,000, but a differential blood count proved negative. At the operation the surgeon found a cyst-like globular mass, the size of a child's head, situated in the position of a distended bladder. The bladder itself could be felt low down in the pelvis to the right of the tumor. The latter was free on all its surfaces except on the left, where it appeared to be adherent to the lateral pelvic wall. There was no connection between the tumor and the colon, sigmoid, or rectum. On aspiration 22 grams of an odorless, puriform fluid was withdrawn. This was reported to contain vacuolated organisms. The cyst-like tumor was opened and then packed with iodoform gauze; the edges were sutured to the abdominal wall. Cultures from the contents remained sterile. The patient made a good recovery and left the hospital with a discharging sinus. Slight weakness and some loss of weight followed the operation; otherwise, he was well. The discharge lessened, his health improved, and, when he came to our clinic, he was feeling quite well. The discharge from the sinus resembled vaselin. There was some resistance in the region of the scar, and an indefinite mass, not tender on pressure, could be felt. The urine was clear, acid, with specific gravity of 1012, and was free from albumin, sugar, and sediment.

Under ether we packed and closed the sinus, made an incision a little to the left of the median scar through the posterior sheath of the rectus and entered at once into a cyst. This contained a vaselin-like substance enclosing minute, brownish dots of the size of pinheads that resembled blood, masses of brightly refracting, yellowish granules, and some odorless, watery fluid. The cyst wall was 2 to 3 mm. in thickness and lined with a membrane much like that of the bladder. The wall, though friable, could be peeled from the surrounding tissues by sponge dissection. As the tumor was freed more and more it was found to lie entirely without the peritoneum, and to be situated close beneath the median line and somewhat to the left. As the deeper portions were drawn upward into the wound, the cyst was seen to terminate in a cone-shaped process, from which, without a visible canal, a definite cord, 5 mm. in diameter and about 4 cm. long, passed to the bladder, apparently at the latter's fundus. On the supposition that we were dealing with a urachal cyst, the cord or tube was ligated and cut and the sac turned upward and forward from below. Above, it extended in a pouch-like process to the region of the umbilicus, and this portion, having been dissected up rapidly and carelessly, the cyst was removed. As soon as tension was removed sharp arterial hemorrhage came from the deeper part of the abdomen, and the original abdominal incision was rapidly extended upward. The source of the bleeding proved to be an arterial branch of the sigmoid flexure. The latter was found to have been cut across as it lay against the anterior abdominal wall. The upper angle of the abdominal wound opened for the first time the abdominal cavity, and the sigmoid was found to be lying obliquely at the level of the umbilicus and pushed forward against the anterior abdominal wall by the cyst, which had been posterior to it and behind the peritoneum. The cavity previously occupied by the cyst extended from the median line anteriorly to the median line posteriorly, pushing down into the pelvis behind the bladder, which was forced upward and to the right. With the restoration of the bladder to its normal situation, the cordlike termination of the cyst was seen to be attached to the bladder at the normal insertion of the left ureter, and examination of its stump showed that it contained mucous tissue centrally. Careful search for a proximal ureteral stump proved fruitless. There was no leakage of urine from the left renal region, and massage of the left kidney, which was normal to palpation, failed to bring any urine into the field of operation. The accidental opening of the sigmoid was closed with a double row of linen sutures, and, so far as could be seen, there was no evidence of disturbance in its blood supply, although, in the light of later developments, and judging from our previous experience in dealing with the colon and sigmoid, we believe that there was sufficient interference with its blood supply to cause a limited necrosis. The abdominal wound was closed, allowing room for a cigarette drain to the pelvis. Owing to possible infection from the opened gut, we did not explore the right kidney region, but later *x*-ray examination established without question the presence of a normal kidney on that side. The patient made a good recovery, and was sitting up when, on the thirteenth day, a small fecal fistula developed; this closed spontaneously after several weeks. Six months after operation the patent was well.

The pathological report made by Dr. W. T. Councilman, of the Pathological Department of the Harvard Medical School, is as follows: The specimen was received, cut up, and placed in Zenker's and formalin solutions. It consists of a thick-walled, sacculated cyst, probably about 7 to 9 cm. in diameter. The inner surface is brownish in color and velvety in appearance. The partitions between the recesses are thick, with smoothly rounded internal borders. Two pieces contain each a funnel-shaped depression, terminating in a short, thick-walled tube of small calber. The cyst throughout is lined with stratified epithelium similar to, but not exactly repeating, the epithelium of the kidney pelvis. In some places there is but a single layer of cells; in others, a considerable degree of thickening. Back of this epithelium, throughout all the sections, there is a reticular tissue enclosing cells corresponding to small lymphoid cells. In places these cells are aggregated in masses resembling small lymph nodes. Similar aggregations of lymphoid cells are found scattered throughout the tissue at varying distances from the internal surface. The general mass of the cyst wall is composed of dense, fibrous tissue, in which masses of non-striated muslce fiber are embedded. On the interior of the cyst,back of the reticular tissue, the muscle forms an almost continuous wall. Elsewhere the muscle appears in irregular masses, cut at various angles. In the outer portion of the thinner parts of the cyst wall there are numerous hyaline, fibrous masses, in all respects resembling hyaline, obliterated glomeruli. In these places there are a number of ducts, varying in size, and lined with a layer of small cells, with well-staining nuclei. Sometimes the lumen is free; at other times it is filled with hyaline material. In the thicker portions of the wall the tissue is composed of easily recognized renal tissue. In this there are numerous glomeruli, separated from one another by masses of tubules, cut at various angles. In these places the amount of connective tissue between the tubules corresponds to that of the normal kidney. The tubules, however, do not show the normal differentiation. Most of them are lined with a low, rather irregular, epithelium. Here and there, however, tubules can be found with an epithelium corresponding to the convoluted tubules. There are, also, numbers of rather small tubules with an epithelium corresponding to the convoluted tubules, and here and there the Henle tubules can be recognized. In most of these places the lumina of the tubules are free. This tissue corresponds to that of the normal kidney, except that the glomeruli are relatively fewer in number and there is not the same differentiation in epithelium. Throughout the wall in all the sections there are great numbers of thick-walled blood-vessels, both arteries and veins, and large numbers of small nerve bundles. The whole structure closely corresponds to a hydronephrotic kidney with great atrophy of renal tissue and, in places, with tubular hyperplasia.

There are various types of anomaly of the kidney, some of which, such as absence of one kindey, horseshoe kidney, fusion, double pelvis, etc., are well known and recognized in the living. A supernumerary kidney, however, appears to be much less common, and on this account it seems worth while reporting our case.

In 1904 Pohlman,[1] of Baltimore, in his article on "Embryology of Kidney Anomalies," suggested a satisfactory explanation for the development of supernumerary kidneys, although he did not report an illustrative case of his own.

Normally, in the development of the embryo, the renal bud arises dorsally on the Wolffian duct after the duct has reached the cloaca, and at a short distance from its point of entrance. The Wolffian duct is of mesodermic origin and is the first portion of the excretory system to make its appearance. The buds, two in number, grow dorsally and lie close together. each being capped by a specialized mesodermic tissue, the renal mesenchyme. Each bud divides into an upper and a lower sprout at some distance from the Wolffian duct, and the two sprouts go to form one kidney. The kidney wanders upward from a position in front of the second sacral vertebra, rotates at the midlumbar region, and finally reaches its normal height at about the end of the second month. The kidney becomes vascularized after it reaches its normal height. In the development of supernumerary kidneys (which occurs before the third month) each sprout on one side or the other, or on both, is surrounded by an independent mass of mesenchyme, forming one or two supernumerary kidneys, as the case may be.

According to Quain, the occurrence of a third kidney is extremely rare; it is placed either in front or on one side of the vertebral column or in the pelvic cavity. Sometimes the third kidney may be united to one of the others.

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[1] Amer. Med., 1904, vii, 987.

The following cases bearing on this subject have been reported:

Newman,[2] in 1898, had seen only one instance of a supernumerary kidney. That was found at a postmortem examination on a male subject who had died from malignant disease of the colon. It consisted of a small, pear-shaped body lying close to the upper margin of the left kidney; its blood supply was furnished by a branch of the renal artery. It had a distinct ureter, which passed into the left ureter half an inch below the pelvis of the left kidney. Sections of this mass examined microscopically showed healthy renal tissue. The mass was completely distinct from the kidney proper, being united to it only by the little ureter.

Debierre[3] related a case, recorded by Gavard, in which three kidneys were situated against the lumbar spine, the ureter of the central kidney opening into the ureter of the right kidney. He thinks that this was a case of complete division of the right kidney. No further particulars are given.

In 1899 Cheyne[4] reported a case of movable third kidney. An hysterical woman, 22 years of age, complained of indefinite abdominal pain and indigestion. During the attacks of pain she noticed a diminution in the quantity of urine passed. On physical examination, a tender swelling was found below the umbilicus, behind the right rectus, at the brim of the pelvis; irregularly flattened, nodular, and somewhat, but not freely, movable. It could not be felt by pelvic examination. At operation the tumor was found to be retroperitoneal, resting on the right side of the lower lumbar vertebræ and on the brim of the pelvis. When exposed, a normal, lobulated kidney presented. Both flanks were examined carefully and normal kidneys were found on both sides of the vertebral column, the left being somewhat smaller than the right. The well-developed third kidney had its own ureter and blood supply and lay about three or four inches below the right kidney. It was replaced without fixation, as the tearing necessary to expose it would, it was thought, cause adhesions enough to hold it in place. The patient made an uninterruted recovery and reported herself at the end of a year relieved of all her symptoms. A diagnosis of the condition was not made before operation. The question of a movable kidney was ruled out on account of the position of the tumor and the fact that it could not be pushed into the loin. The symptoms were thought to be due to the mobility and to the slight pressure on or kinking of the ureter.

Bartlett[5] reported a case of supernumerary kidney under the

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[2] Malformations of the Kidney, Clin. Soc. Trans., 1898, xxxi, 118.

[4] A Case of Movable Third Kidney, Lancet, 1899, i, 215.

[3] Ibid.

[5] A Pathological Curiosity (Supernumerary Kidney), Lancet, 1904, i, 124.

heading, "A Pathological Curiosity." At an autopsy two kidneys were found on the left and one on the right side. The two on the left were fused together, one being above the other. The two ureters on the left arose separately from two distinct pelves and continued separately to the bladder, one entering the bladder one-quarter of an inch above the other. On section a distinct demarcation of the two kidneys was not visible to the naked eye. All three were normal in appearance. They had no connection with the cause of death. He states that a supernumerary kidney is very rarely met with. He regards it as an extreme instance of segmentation of the kidney. The most typical arrangement is in serial order with the kidney on the same side, but entirely separate. It has its own vessels and duct, but the duct may be fused with that on the same side or remain separated.

Tschudy[6] in 1902, reported a case of complete double formation of one kidney; the upper portion was formed by a separate pelvis, which was distended and septic; this emptied into a thick, infected separate ureter; the lower half was formed by a normal kidney, with its normal ureter. The parenchyma in the upper half had disappeared, because of the pressure of the pelvic abscess; it showed microscopic remnants of parenchymal substance at the hilum. The stenosis of the ureter lay at the vesical end. The patient survived operation.

TENDON TRANSPLANTATION AND NERVE ANASTOMOSIS.

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TENDON TRANSPLANTATION.

Tenoplasty, or tenontoplasty, according to Hoffa,[1] of Berlin, was first conceived and employed by Nicoladoni, in 1882, and has now become one of the most important operations in the whole field of orthopedic surgery. This operation in latter years has been much talked about, although it was invented and tried in single cases at a much earlier period. Arthur W. Elting,[2] in an extensive review of the subject, states that Duplay, in 1876, transplanted tendons. A short history of the operation may be quoted from H. Augustus Wilson,[3] who says, " that in 1892 Parrish, of New York, reported a case, the first in America. The same year, Drobnik proposed periosteal insertion of transplanted tendons. In 1894, Winkelman published and analyzed 16 cases. Since 1894, Goldthwait, of Boston, and others have perfected the methods. In 1898, F. S. Eve reported 274 operations, and Vulpius, in 1899, gave his experience in 160 cases. In 1902, Lange introduced artificial silk tendons, following the example of Glück, who, in 1892, had used bundles of silkworm gut to bridge the gap, after division of tendons in injuries. Kummell, in 1896, found that the strands ultimately became converted into fibrous tissue."

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[6] Ueber einen Fall von Doppelbildung der linken Niere mit Pyonephrose des einen Nierenbecken Harnleitersystems, Cor. Bl. f. Schweiz. Aerzte, 1902, xxxii, 400.

[1] Amer. Jour. Arthop. Surg., August, 1904.

[2] Albany Med. Jour., April, 1902.

[3] Amer. Med., April 8, 1905.

The indications for the operation are as follows: Cases in which function is faulty, because of ill-directed voluntary movements, aiming to restore the balance of power as far as possible; in traumatic loss of function of muscles and tendons; spastic paraplegia; infantile paralysis, and congenital deformities.

Vulpius,[4] of Heidelberg, says that tendons may be transplanted in three ways: (1) We may simply freshen the paralyzed and healthy muscles at the sides and sew them together. This method is rarely used, because of the weakness of the scar. (2) We can unite a healthy tendon as a whole with a paralyzed one, or total transplantation. This is only done when the sound muscle has an unimportant function. The peripheral portion of the sound tendon is then attached in an upward direction to a neighboring tendon of similar function. In this way, the function of the sound muscle is not lost. (3) We may take a slip from a sound tendon and insert it in a paralyzed one. The paralyzed tendons may be treated in three ways: (1) We may cut them entirely and turn them upward to the sound tendons; (2) we may divide them and attach a slip to the healthy tendon; and (3) we may leave them intact and unite them in some way to the sound tendon.

The old operation, as performed by Nicoladoni, united tendon with tendon. A modification of this has been instituted by Lange, who does a periosteal transplantation, that is, he unites the sound tendon to a point on the periosteum of the bone to which the paralyzed tendon is attached. He claims two advantages for this method. He thinks that with the old method the paralyzed tenddon will stretch, thus causing insufficient tension and failure; and further he claims a free choice of insertion for the tendon. Some surgeons, notably Vulpius, do not agree with this view. They claim that the paralyzed tendon does not stretch, and that Nature's insertion of tendon cannot be improved upon. Wolff places the tendon in a bony canal, and Muller has even conducted a tendon entirely through a bone. In order to insert tendons in the periosteum, they often have to be lengthened. This necessitates a lengthening by silk strands, thus making a false silk tendon. This was recommended and successfully used by Glück. The threads afterward become enclosed in a fibrous deposit which is mixed up with tendon fibers until finally a new tendon is built up with a central core of silk threads. According to experiments made by Lange, we know that the young tendon tissue creeps along the silk threads, as along a lattice. He has shown that in sufficiently old artificial tendons, the tissue penetrates into the inside of the silk threads. There is an interlacing, so to speak, and finally the silk threads lie embedded in the newly-formed tendon. There has also been observed a condition in which only a connective-tissue regeneration takes place, and there is formed a kind of tendon sheath in which the silk glides, as the working tendon. The most important duty of the silk tendon is to retain the body of the transplanted tendon at a proper tension, to prevent its retraction and shrinkage, until the young tendon has gained sufficient firmness to undertake the work itself. The tendon sheath is not regenerated, therefore it should be spared as much as possible in operating. The tendon preserves a grayish-white color, a sign of a more scar-like constitution. There are additional tendinous bands formed from irritation and extension of the inflammation; but, on the whole, the transplanted tendon heals into position perfectly without much reaction in itself.

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[4] New York Med. Jour., September 10, 17, 24, and October 1, 8, 1904.

An important point is the amount of tension to be obtained: too much injures the muscles, while too little interferes with the action of the tendons. When tendons are carried for some distance for the purpose of transplantation, tunnels must be made for them through the tissues with a blunt instrument. These tunnels or canals should be as straight as possible, as an angular tunnel interferes with the power of traction. Results are better and are seen sooner if the transplanted tendon is kindred in function to the paralyzed tendon. In order to achieve good results, four conditions are necessary, namely, (*a*) perfect asepsis; (*b*) prevention of hemorrhage; (*c*) healthy muscle material, and (*d*) union under certain tension.

The paralyzed muscle, says Koch, undergoes more or less fatty degeneration, and these fatty deposits occur in continuity or at intervals. It is noteworthy that accompanying this fatty degeneration there is always a regeneration taking place. Upon this fact rests the recovery of the paralyzed muscle. We can transplant with success only such muscles as possess some power of regeneration. The American surgeon, as a rule, uses only red or pink muscle, but the Germans will use yellowish-red or even quite yellow muscle, on the ground that they do at times ultimately regenerate. It is of the greatest importance that the muscle ransplanted be united with the other tendon under sufficient tension. In anterior poliomyelitis, both the elasticity and the tone of the muscle are lost, but there still remains the power of regeneration. This alone, however, does not suffice to bring back the muscle tone. We must, therefore, by the operation restore the elasticity and tone, and this can only be done by shortening the muscle itself. This is the essential factor leading to good results. The word tension is here used in the sense of overcorrection, and not strain.

Hoffa has done some original work in tendon grafting. He has transplanted the trapezius to the position of a paralyzed deltoid in shoulder-joint paralysis. He has changed the action of the pronator radii teres by transplanting it from the internal to the external condyle of the humerus, and thus overcome the pronation of the forearm. Muscles and tendons that have had their function changed from extension to flexion, pronation to supination, and vice versa soon work very well in their changed order. The brain cells become accustomed to the new order of things in a surprisingly short time. If the mental intelligence of the patient be good, the functional result will be better than if the mental condition be of a low grade.

In 1902 James K. Young and James A. Kelly,[5] of St. Mary's Hospital, Philadelphia, reported 2 cases. In the first case, one of talipes calcaneus, following infantile spinal palsy, the tendon of the peroneus longus was implanted into a paralyzed tendo Achillis. The latter tendon was divided obliquely; its two cut ends were then shortened and stitched together with interrupted fine silk sutures, except at a point in the middle of the cut. Through this opening the tendon of the peroneus longus was carried and stitched in place with interrupted sutures of fine chromicized catgut. The sheath of the tendo Achillis was then stitched together and the wound closed. Ten weeks after the operation, the brace worn was discarded and the patient showed very good results. In the second operation the tendon of the extensor proprius hallucis was implanted in the tibialis anticus, for talipes equinovalgus. Good results followed after two and a half months.

In 1903, Royal Whitman,[6] of New York, called attention to the importance of supplementing tendon transplantations by other procedures designed to assure stability and to prevent deformity, otherwise, the results would be on the whole disappointing. It is, of course, evident that tendon transplantation should be most effective in the slighter grades of paralysis. It is particularly indicated in paralysis of the anterior leg muscles, with consequent lateral distortion of the foot and slight toe drop. Whitman has performed the following operation for such a condition: First, an arthrodesis of the astragulonavicular articulation, that is, the cartilage is removed from the opposing surfaces; then the extensor proprius hallucis is divided near the great toe, drawn through a hole bored in the navicular and sutured to itself and the periosteum, to insure sufficient tension and hold the foot in the normal position.

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[5] International Clinics, 1902.

[6] Amer. Jour. Orthop. Surg., August, 1903.

The tibialis anticus is shortened to the same degree, and the tendo Achillis is divided if necessary. The foot is then placed in a position of slight adduction and put up in plaster-of-Paris for two months or longer. The patient uses the foot in walking as soon as possible.

In paralysis of the calf muscles, the peroneal tendons are attached to the os calcis after arthrodesis of the ankle-joint. In paralysis of the peroneus brevis, causing varus of the foot, the extensor hallucis should be attached to the cuboid bone, and the foot fixed in abduction.

In 1904, John Dane and David Townsend, of Boston, reported the after-results of fifty tendon transplantations. The majority gave poor results, but this was probably due to a poor selection of cases.

Dr. Hans Spitzy,[7] of Graz, Austria, reports a case in which a new tendon was formed for the hamstring tendons. A silk tendon had been made, and in three years it was as large as the finger. In one case, in which the tendon was too long, a piece was removed and examined microscopically. There was found to be an intertwining of the fibers.

In April, 1906, Phil Hoffmann and Sydney I. Schwab, of St. Louis, reported an interesting operation for cerebral palsy, involving the flexors and extensors of the forearm, dating from a few days after birth. There was good power in the upper arm. The insertion of the biceps into the radius was cut away and it was inserted into a longitudinal slit in the bellies of the palmaris longus and the flexor carpi radialis, which had been previously detached from the inner condyle of the humerus. These muscles were paralyzed and were simply used as passive structures because they were long and slender and acted as a long tendon. The tendons of these two muscles were cut from their insertion at the wrist and sewed en masse to the tendons of the deep and superficial flexors, including the thumb. The pull of the biceps is now transmitted through these two muscles to the fingers. The patient can hold a fork and spoon, and can lift a five-pound flat-iron and hold it for thirty seconds. He wears a splint to hold the wrist in an extended position, so that his new flexors can act to good advantage. An arthrodesis of the wrist will later be performed.

Much of the success of tendon grafting depends on the after-treatment. After such an operation, the limb should be immobilized in a plaster-of-Paris cast from four to six weeks. After the removal of the cast, the part should be enclosed in splints for six months. During this time, massage, electricity, gymnastic exercise, and baths should be made use of. The longer and more careful the after-treatment the better are the final results.

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[7] Med. Fortnightly, April 10, 1906.

Tendon transplantation is not a cure-all. When Nicoladoni's enthusiasm reached the clinic at Vienna much work was done, but it was quickly abandoned, probably because the operation was looked upon as largely curative, and no supplementary measures or apparatus were then used. It is the combination of procedures, taken in connection with the transplantation, which gives the best results.

NERVE ANASTOMOSIS.

Nerve grafting is a step higher than tendon grafting. It is undertaken in order to restore function in incised or paralyzed nerves. There is some doubt as to who first performed the operation. In 1894 or 1895, Charles F. Smith[8] first reported an operation on two horses for laryngeal paralysis. In both cases the proximal end of the spinal accessory was sutured to the distal end of the laryngeal recurrent, with beneficial results to both horses. In 1897, Dr. G. Carl Huber published some experiments on "Nerve Suturing and Nerve Implantation," which gave some improvement in technique. In 1898, Frank E. Peckham,[9] of Providence, R. I., transplanted two branches of the internal popliteal into the external popliteal for paralysis of the extensors of the leg. He says that partial power was restored, but fails to give any details. A second case of like character resulted in a very slight movement of the toes at the end of six weeks. In 1902, Harvey Cushing[10] operated on a case for traumatic facial paralysis. The facial nerve was anastomosed with the spinal accessory. This nerve was divided entirely, thus sacrificing its motor territory. In five months the patient had recovered some use of the facial muscles, at first not coördinated, but afterward they became so. In nine months the electrical response on both sides of the face was practically the same. Cushing says that the first case was operated on by Faure, in 1898, after traumatic injury to the facial nerve. It was unsuccessful, probably owing to the long duration of the injury. The second case was that of Kennedy, who intentionally divided the facial nerve for spasmodic tic, and made an anastomosis, with the same results as those of Cushing.

In 1902, W. W. Keen,[11] of Philadelphia, performed a similar operation at the Jefferson Hospital. He anastomosed a section of the spinal accessory 3 cm. long with the facial, for facial palsy following trauma. Three months afterward the condition was unimproved.

The first case of nerve anastomosis for infantile spinal palsy was performed by James K. Young, of Philadelphia, at the Polyclinic Hospital, in December, 1902.[12] It was reported in February, 1903, before the Philadelphia Neurological Society, and afterward at the annual meeting of the American Medical Association. In this instance the tibialis anticus muscle alone was paralyzed. The nerves supplying this muscle were inserted into the musculocutaneous. Decided improvement followed, and the patient is now able to walk almost without a limp and she can turn her foot in, which before was impossible.

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[8] Brit. Med. Assoc., 1894 or 1895.

[9] Providence Med. Jour., January, 1900.

[10] Jour. Mental and Nervous Dis., June, 1903.

[11] Amer. Jour. Orthop. Surg., August, 1904.

[12] Jour. Amer. Med. Assoc., January 21, 1905.

About two months after this operation Charles H. Frazier,[13] of the University of Pennsylvania, performed a similar operation, with not quite so marked results, but still some improvement was noted. In this case the musculocutaneous was grafted into the anterior tibial nerve.

In 1904, 3 cases were reported by Alfred S. Taylor and L. Pierce Clark,[14] of New York, for facial palsy. They anastomosed the facial and hypoglossal. In March, 1906, they again reported the cases, both one of which had developed motor power on the paralyzed side.

A later operation by James K. Young was reported on May 7, 1906, before the Academy of Surgery of Philadelphia. The external popliteal was grafted on the internal popliteal, for paralysis of the peroneal muscles. In this case sensation was restored in twenty-four hours, and some improvement in the motor power has already been noted.

An operation for quadriceps paralysis has been proposed by Dr. Hans Spitzy.[15] He suggests inserting the long branch of the obturator into the anterior crural nerve.

There are three methods of transplanting nerves: (1) A slip from a sound nerve is inserted in a paralyzed nerve—or central transplantation; (2) the paralyzed nerve may be divided and the distal end inserted into a slit in a sound nerve, or peripheral transplantation of the total central type; (3) a slip of a paralyzed nerve may be inserted in a slit in a sound nerve, or peripheral transplantation of the partial central method. The technique requires all aseptic precautions, and its success depends on the use of small instruments (eye instruments being preferred), and as little handling of the nerve as possible. The distal end of the divided or paralyzed nerve should be secured in a longitudinal slit in the sound nerve by means of several catgut sutures attached, if possible, only to the nerve sheaths. The transplanted nerve should be so placed that its central portion is in contact with the central portion of the sound nerve, and looking in the same direction. The proximal end of the paralyzed nerve should be inserted in a neighboring muscle, in order to prevent the formation of a neuroma. The separation of the sheath is possible in large nerves, but not in the smaller ones.

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[13] Med. Record, February 27, 1904.

[14] Jour. Amer. Med. Assoc., March 24, 1906.

[15] Amer. Jour. Orthop. Surg., August, 1904.

Taylor and Clark, of New York, recommended wrapping the junction of the two nerves in cargile membrane to prevent the ingrowth of connective tissue to the field of operation. The wound is then closed without drainage, and the part put up in plaster-of-Paris for from four to six weeks. Afterward, electricity and voluntary movements are cautiously begun. It is said by recent observers that electricity stimulates the fibrillation of the axis cylinders in which consists the final stage of nerve regeneration.

In anterior poliomyelitis, when only one or two muscles remain paralyzed, the operation should be performed after a lapse of about six months. It is not permissible within three or four months after the acute attacks. It is the opinion of most orthopedic surgeons that those muscles which still remain paralyzed after six months are not likely to regain their power. The operation is best suited to those cases in which a few or only one muscle remains paralyzed. Some nerve cells in the anterior horns are so seriously damaged that they are incapable of regeneration. Others are only diseased, and are still capable of partial regeneration, and function will return later to the muscles innervated by these cells. The muscles that remain permanently paralyzed represent nerve cells which have degenerated beyond repair. It is the usual experience that restoration of function occurs in some of the muscles which at first were partially, or even completely, paralyzed. This takes place in a few months after the onset. When only one or two muscles are paralyzed we may expect good results by uniting the nerve supply of these muscles with a motor nerve in the vicinity, as in this way healthy nerve cells are brought in contact with diseased nerve fibers, and the latter may regenerate. The ideal method of anastomosis would be to place the axis cylinder of the divided nerve in relation to and parallel with the axis of the sound nerve.

There are two theories of nerve regeneration: (1) The axis cylinder of the sound nerve grows peripherally into the degenerated nerve. When the centre is re-established, the muscles regenerate, and are again able to do their work, since their degeneration was secondary to that of the nerve. (2) The regeneration takes place from the sheath of Schwann. This is not generally accepted.

According to Spiller, the later investigators have shown that the peripheral stump takes part in the regeneration. It is probable that this regeneration is not permanent unless there is union with the central stump. In answer to the question of how to get proper coördination when the peripheral anatomy is so disturbed, Dr. Spitzy says that the seat of coördination being in the brain, it is possible, when a peripheral nerve is paralyzed, that another centre may take on the function of the paralyzed one. There are many ways for the impulses to come from the different centres, and it is possible for the centres to be re-educated to the new work which they have to perform. Dr. Harvey Cushing adds the theory that new centres may be developed in children, but not in adults. There we have an education of the old nerve centres to do the different work.

When the operation is successful, decided advantages will have been gained over tendon transplantation. In the latter the muscles that have been diseased are thrown out of function entirely, and use other muscles to perform a function that heretofore they have not been called upon to do. Nerve anatsomosis restores the function of the paralyzed muscle and leaves all other muscles intact.

The future of the operation can only be speculated upon. The careful selection of cases is the most important element of success. However, from results already obtained we cannot but feel that the future has probably in store for us many surprises which may lead to gratifying results, especially in the treatment of anterior poliomyelitis.

THE PLEURAL PRESSURE AFTER DEATH FROM TUBERCULOSIS.

A PRELIMINARY STUDY.

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The existence of a minus or negative intrathoracic pressure was first noted by Carson in 1819. The subject was later discussed by Donders,[1] who established the figures for this pressure and gave the variations in pressure occurring with inspiration and expiration. Hutchinson, Heynsius,[2] Perle and others have made investigations along this line, so that the existence of a negative intrapleural pressure may be considered as proved. This negative pressure is due to the fact that the lungs are constantly distended and the elastic fibers of the lungs are constantly striving to regain a condition of non-tension. Hence, the amount of intrapleural pressure under normal conditions would show the amount of the elastic tension of the lung. This distention of the lung and its tendency to become smaller is well shown by the collapse of the lungs when the chest is opened, either during life or after death. It is because of this condition of a negative intrapleural pressure that a pneumothorax occurs when the lung ruptures or the chest is perforated from without.

Despite the many publications upon the subject only one reference could be found which attempted to explain the origin of the intrapleural negative pressure. Landois[3] states, "that until birth the airless lungs lie collapsed (atelectatic) in the chest cavity, and fill it so that pneumothorax is not produced if the thorax be opened in a dead fœtus. Even in children who have lived for eight days and breathed normally, the lungs do not collapse when the pleural cavity is open, but remain in contact with the chest wall. It is only after further growth that the thorax becomes so large that the lungs must expand under elastic tension; only then will opening of the thorax cause the lungs to retreat into a smaller volume." This explanation seems to be in accord with the anatomical and physical conditions present. The theory of West that the layers of pleura are held together by cohesion are as two panes of wet glass does not seem tenable.

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[1] Physiologie des Menschen, Leipzig, 1859.

[2] Archiv f. d. gesammte Physiologie, 1882, xxix, 265.

[3] Text-book of Human Physiology, Blakiston, 1904.

There are several methods by which the intrapleural pressure can be studied. Thus, Donders, Hutchinson, Perle, and others connected a manometer with the trachea of the cadaver, and measured the pressure produced by opening the chest cavity. This pressure was, of course, positive, and showed the elastic tension of the lungs. Likewise it was a measure of the negative pressure within the pleura. The pressure thus found is supposed to represent the pressure that obtains at the end of quiet expiration. Donders believed that beside the elastic tension, which is measurable after death, there existed during life a condition of pulmonary tone which made the lung tension, and accordingly the negative pleural pressure greater. To prove this he fastened a tube connected with a manometer in the trachea of a healthy animal and killed it by suffocation. As soon as the manometer was at zero the chest was opened, and the pressure from the lung contraction noted. The manometer was left attached and after a short interval of time it was noted that the pressure had diminished one-fourth. This loss in pressure Donders thought to be due to the disappearance of the pulmonary tone. In eleven cadavers examined by him he found a pressure varying between 30 and 70 mm. of water. As the lung in all these cases showed more or less disease, Donders assumed the normal lung would show a greater pressure. He estimated that the normal lung at the end of expiration would show a pressure, due to elastic tension alone, of 80 mm. of water. To this must be added one-fourth, or 20 mm. water, due to pulmonary tone. Thus, according to Donder's estimate, the normal healthy lung in life would show a tension of 100 mm. water, or 7.5 mm. Hg., at the end of quiet expiration. Quiet inpiration increases this tension to 9 mm. Hg., and forced inspiration probably to 30 mm. Hg. While these figures of Donders are quoted in all text-books, it will be noted that they are not based upon experimentation. This method was carried out by Perle,[4] who estimated the pressure in 100 cadavers.

Adamkiewicz and Jacobson[5] attempted to measure the intrathoracic pressure by inserting a cannula into the pericardium, while Heger and Spehl[6] made a pericardial fistula through which they studied the pressure in rabbits. Luciani and Rosenthal[7] estimated the pressure by means of a sound inserted in the œsophagus. D'Arsonval, in 1877, and Fredericq, in 1882, measured the pressure by inserting a hollow needle into the pleural cavity. The majority of these experiments were done on animals. Because of the disturbed respiratory rate from the attachment of the apparatus, the results on living animals were found to be untrustworthy. Therefore, even the figures given of animals are not those present during life. In the human being during life puncture of the interspaces is the only possible way to learn the intrathoracic pressure, and because of that this method was adopted in the present study.

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[4] Archiv f. klin. Med., 1869, vi, 1.

[5] Centralbl. f. d. med. Wissenschft, 1873, 483.

The observations here reported were made upon subjects after death at the Henry Phipps Institute as a part of the study of the pleura and its behavior in tuberculosis. The method employed consisted in the insertion of a hollow needle, which was connected with a mercury manometer, into each of the five interspaces. This was done before the body had been touched by the pathologist. All examinations were made within twelve hours after death; one or two while the body was yet warm. When the pressure was negative the needle was pushed still further into the chest to find, if possible, the pressure condition within the lung. The punctures were made anteriorly along the anterior axillary margin. The skin was pulled as far as possible to one side and the pressure made through the tense tissue. The return of these tissues to a natural position acted as a valve to prevent ingress of air after the needle was removed.

The results here given will have to stand by themselves, as it has been impossible to find a record of a similar investigation. Besides, the result of the pressure study it has seemed advisable to give also a tabulated list of certain deformities, such as spinal curvatures and displacement of the heart, and the symptoms of pain and dyspnœa.

The total number of cases examined was 23. Of these, 17 were males and 6 females. As to age, there were 2 between fifteen and twenty; 5 between twenty and thirty; 8 between thirty and forty; 7 between forty and sixty; and 1 of sixty-three.

Pain in the chest was present in 14; absent in 7; not noted in 2. Dyspnœa was present in 18; absent in 4; not noted in 1. Spinal curvature was present in 7; absent in 14; not noted in 2. The apex beat of the heart was outside the midclavicular line in none; in the midclavicular line in 13; inside the midclavicular line in 8; not noted in 2. Retraction of the left chest anterior was present in 9; absent in 11; not noted in 3. Retraction of the right chest anteriorly was present in 8; absent in 13; not noted in 2.

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[6] Archiv de biologie, 1881, 153.

[7] Archiv f. Physiologie, von du Bois-Reymond, 1880.

The frequency with which depression is found above and below the clavicles is too well known to require comment. It was likely that it was present in every case. Our former history records this fact under the heading of, "Difference in Depression above Clavicles." This was greater in the right in 10; on the left in 1; equal in 10; not noted in 2.

The conditions found at autopsy are of especial interest. The two important things in the present study were height of the diaphragm and the extent and distribution of the pleural adhesions. In a later communication the condition of the lung will be dealt with. The diaphragm was found at the fourth rib once on the left; at the fourth interspace 4 times on the right, and 4 times on the left; at the fifth rib 9 times on the right, and 7 times on the left; at the fifth interspace 8 times on the right, and 7 times on the left; at the sixth rib none on the right, and twice on the left; at the sixth interspace 1 on the right, and 1 on the left; at the seventh rib 1 on the left. In 1 case the pleura was adherent to the diaphragm on the right side, and the level was not obtainable. It will be noted that in the greater majority of cases the diaphragm was found between the fourth and the fifth interspaces. This was the condition in all instances on the right side except 1—a case of pneumothorax. On the left side in 18 cases the diaphragm was between the fourth and the fifth interspaces. In the 2 left-sided pneumothoraces and 1 right-sided pneumothorax it was below. In 1 instance it was found at the fourth rib on the left.

The number and distribution of the pleural adhesions were as follows:

The left lung showed adhesions of greater or less extent over the upper lobe in every instance. The left lower lobe was more or less adherent in 8 cases; free in 15. Over the right lung the upper lobe showed more or less adhesion in 21 cases; free in 2; right middle lobe adherent in 11 cases; free in 12; right lower lobe adherent in 10 cases; free in 13.

In this series the upper lobes of both sides showed pleural adhesions to be practically constant, while in more than one-half of the cases the lower lobe was free.

The results of the puncture of the interspaces were as follows: On the right side the first interspace gave a minus pressure in 8; a plus pressure in 3; a zero pressure in 11 out of 22 cases. The second space gave a minus pressure in 10; a plus pressure in 2; a zero pressure in 11. The third space gave a minus pressure in 14; a plus pressure in 2; a zero pressure in 7. The fourth space gave a minus pressure in 14; a plus pressure in 3; a zero pressure in 2.

It will be noted that as we go down the chest the number of cases showing a minus pressure steadily increases, and at autopsy it was found that the number of adhesions decreased. The same conditions were generally present on the left side where the results were as follows:

The first space showed a minus pressure in 7; a plus pressure in 3; a zero pressure n 13. The second space showed a minus pressure in 7; a plus pressure in 2; a zero pressure in 14. The third space showed a minus pressure in 9; a plus pressure in 3; a zero pressure in 11. The fourth space showed a minus pressure in 13; a plus pressure in 2; a zero pressure in 8. The fifth space showed a minus pressure in 18; a plus pressure in 1; a zero pressure in 3.

The majority of cases with obliterated pleura showed a zero pressure when the puncture was made through the adhesion. In a few instances a minus pressure was obtained. This apparent discrepancy was due, in all probability, to a difference in the character of the adhesions. An obliterated pleura may be a complete adhesion of the two layers throughout, or it may be an adhesion consisting of a multitude of attachments which show like a network when an attempt is made to separate the layers. The interstices between this network are of varying size, and if the needle chances to enter one the elastic contraction of the lung will produce a negative pressure.

The cases showing a positive or plus pressure require special mention. In all there were 8 cases which showed a plus pressure in one or more interspaces.

Case No. 4021 was an acute right-sided pneumothorax. This gave a positive pressure of 6 mm. Hg., except in the second interspace where it was zero. The pressure obtained upon the left was peculiar. On tapping the first interspace there was found a pressure of minus 2 mm., which changed to plus 3 mm. when the needle was pushed further inward. In the second space the first pressure was minus 3 mm., changing to minus 1 mm. In the third space the pressure was minus 2 mm., changing to plus 4 mm. Below this the pressure was zero. At autopsy there was found a right-sided pneumothorax. The pleural cavity contained only a very small amount of fluid. The lung was collapsed. A band of adhesion was found in the second interspace where the zero pressure had been found. On the left side the pleura was adherent as far down as the fifth rib.

Case No. 4272 was an acute left-sided pneumothorax. There was a positive pressure of 10 mm. throughout the left side. On the right side the pressure was zero in the first interspace; minus 8 mm. to minus 3 mm. in the second; minus 8 mm. to minus 3 mm. in the third; minus 8 mm. to zero in the fourth; minus 6 mm. to zero in the fifth. No adhesions were present on the right side. The upper and middle lobes showed œdema, anthracosis, and a few scattered tubercles. The lower lobe showed in addition a large area of tuberculous bronchopneumonia at its apex. There was 1000 c.c. of clear fluid in the left chest.

In this connection it may be well to mention Case No. 4630, with left-sided pneumothorax of longer duration. The diaphragm was pushed down to the seventh rib on the left side. A minus pressure of 4 mm. was found in the first space, and minus 2 mm. in the four lower spaces. On the right side the pressure was zero in the four upper spaces, and minus 6 mm. to zero in the fifth interspace; minus 3 mm. to zero in the sixth interspace.

Only 1 case was found with positive pressure throughout both sides of the chest. This was Case 3948. The patient had reached a condition of quiescence when he suddenly died of hemorrhage. The pressure on the right side was found to be plus 4 mm. in the four upper interspaces and plus 5 mm. in the fifth interspace. On the left the pressure was plus 4 mm. in the first space, zero in the second, plus 8 mm. in the third, plus 8 mm. in the fourth, minus 12 mm. in the fifth interspace, which quickly changed to plus 2 mm. as the needle was pushed further in. At autopsy there was found a clot in the trachea, with marked emphysema. Adhesions were present on the right side only over the apex. The left side was completely adherent except at the point where the minus pressure was found in the fifth interspace.

Case No. 5025 showed a positive pressure in the first and fourth interspeces on the right side. Elsewhere, both right and left, a minus pressure was found. As the abdomen seemed unusually distended, the punctures were repeated after the abdomen was opened and a zero pressure was found instead of the plus pressure. At autopsy the pleura was found to be obliterated on the right side, while the left was free, except for a small adhesion at the apex posteriorly.

Case No. 4197 showed a plus pressure of 4 mm. in the first interspace on the left. In the second, third, and fourth interspaces the pressure was zero. In the fifth interspace it was minus 9 mm. On the right side the pressure was zero in the first and second interspaces; minus 6 mm. in the third, minus 8 mm. in the fourth. In the fifth space the pressure was zero, and fluid was obtained.

Case No. 4243 (death from acute pneumonia), showed on the right side a plus pressure of 2 mm. in the second interspace; zero in the third and fourth spaces; plus 3 mm. in the fifth space. On the left there was minus 7 mm. in the second interspace and zero in the interspaces below. At autopsy the right lung was free of adhesions, while the left was adherent to the fourth interspace. The left lung was consolidated practically throughout, while the right was congested with areas of bronchopneumonia. Clinically it was difficult to distinguish this case from one of pneumothorax, because of the hyperresonance and marked subjective symptoms.

The behavior of the minus pressures is interesting when compared with the figures given as normal by Donders. As already stated this author fixed the normal minus pressure at 7 mm. Hg. at the end of quiet inspiration in the living individual. After death he thought it diminished one-fourth, due to the loss of pulmonary tone, or to about 6 mm. In this series of 23 cases a minus pressure of 8 mm. or more was found in one or more parts of the chest in 19 cases. It was found to be minus 16 mm. or more in 8 cases. The greatest negative pressure found was in Case No. 3119, amounting to minus 24 mm. From this it would seem if the figures given by Donders are correct, that the intrapleural tension may be greatly increased locally or generally in pulmonary tuberculosis.

From these 23 cases certain preliminary deductions are justifiable:

1. In subjects dead of tuberculosis pleural adhesions of greater or less extent are practically constant over the upper lobe. The lower lobes are free in about one-half the cases.
2. The intrapleural pressure may vary in the different interspaces of the same chest. This variation is most often due to pleural adhesions which obliterate the pleural cavity at the point of tapping; less often to the lung condition.
3. The intrapleural pressure varies widely in different subjects—from minus 24 mm. in Case No. 3119, to plus 10 mm. in Case No. 4272.
4. The intrapleural pressure may be negative or minus, while the pulmonary pressure is positive or plus (Case No. 3948).
5. The minus intrapleural pressures found in tuberculosis subjects are much increased when compared with text-book standards.
6. Zero pressures are found in one-half the cases in the first two interspaces.

A STUDY OF NORMAL LIVING ANATOMY IN EARLY LIFE.

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AND
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BOSTON, MASSACHUSETTS

Our knowledge of both the normal and the pathological anatomy of human beings has heretofore depended almost entirely on the postmortem findings. These findings, although of great use, especially under normal conditions, have never been so complete as is called for, regarding the different stages of development by both physicians and surgeons in their special work, when they are called upon to diagnosticate these conditions, and in the case of pathological conditions, to treat them.

Still greater difficulties arise when we attempt to determine the true pathology of the various diseases, for in many instances we are dealing only with terminal results which may, by representing the terminal lesions of a number of diseases, especially the different infections, fail to give us the early and truly characteristic lesions of the special disease.

Any means, therefore, by which we can recognize a disease in its early stages, whether it be an effusion in the pericardium or the pleura obscure centres of pulmonary solidification, an osteomyelitis or a clinically hidden fracture, is of extreme value, and, indeed, a necessity, according to our modern ideas of diagnosis for treatment. This means, more or less perfected, has been given us by the Röntgen method, and the knowledge derived from this method, showing us the actual living condition, is becoming more and more extensive and exact.

Like many other methods of diagnosis, however, of which the technique is difficult, discredit has been thrown upon the value of diagnosis by the *x*-rays. This arises not only from the many failures, and, consequently, poor results from lack of skill in carrying out a difficult technique, but also from the almost ridiculous interpretation of the plates by those whose knowledge in no way fits them for this expert work.

There is no doubt whatever that when the technique is carried out, and the Röntgengram read, by experts, wonderful and reliable results can be obtained, giving us an exact knowledge of the internal organs as well as of the skeleton in both normal and pathological conditions.

Granting that the Röntgen method can accomplish this for us, we must allow that a thorough knowledge of living normal anatomical conditions should be acquired before we can rightly understand what we see in pathological conditions and recognize intelligently the lesions of the various diseases during life.

It would be a comparatively simple matter if we could merely study the normal living anatomy of the fully developed adult, and, of course, such a study is necessary and is very important. This living, fully-developed anatomy, however, is merely one link, in fact almost the last one of the much more extensive and complicated chain of evidence which enables us to diagnosticate normal anatmoical conditions through all their stages of development in early life. It is during early life that manifold changes take place continuously, and the younger the individual the more rapid are these changes. A knowledge of the living anatomy of each period of growth for comparison is, therefore, of the utmost importance as the necessary preparation for the recognition of anomalies, of congenital conditions, and of the various diseases. What the study and use of the Rontgen-rays accomplishes is to give us a large part of living anatomy and pathology at either an early or a late period of development of disease, and thus enable us to decide upon the nature of the disease long before, as, in the past, the findings on the postmortem table can be made use of.

Even anatomists know very little regarding the different stages of the many changes which take place in the growth of early life, the appearance and development of the centres of ossification, and the appearance of the zone of proliferation at different ages, yet without this knowledge we cannot intelligently treat many diseases.

In order to aid the practitioner to overcome the difficulty which arises in attempting to memorize the many and rapidly changing anatomical conditions at different periods of development, we have thought it worth while to prepare a series of plates which can be referred to when any especial period of development is being studied for purposes of diagnosis and treatment. Of the many important changes which normally take place in different parts of the body and from which distinct truths can be deducted in studying them by the Röntgen method, probably the most important are those which take place in the zone of proliferation. We merely speak of this as an example of what this method of study may lead to, for it certainly must be far-reaching, and it opens up so many questions of future research that it would be impossible in a paper of this kind to more than suggest them. In this area of ever-changing growth, many infections take place which may greatly affect the future growth and usefulness of special parts.

The various congenital deformities can be especially studied in the epiphyses of the long bones—notably chondrodystrophia. Again, the diseases of nutrition attack the zone of proliferation, notably in rhachitis. Again, how important are the anatomical conditions of these parts in studying the secondary infections which attack them, notably tuberculosis, measles, typhoid fever, and the various pyogenic infections.

The epiphyses are the most important centres of the skeleton, for on them depend its future development, especially in infancy and childhood, and when these centres are impaired there results far-reaching influences in later life. A thorough knowledge of the different changes which take place in even this small area is very important, for these centres are where disease may begin, from which disease may disseminate, and where it may lead to disastrous consequences and may leave its permanent marks.

Very little is known of the etiology of the diseases of nutrition and the especially important rhachitic conditions which play such a great role in early life, and we also have a comparatively slight knowledge of its early stages. A more exact knowledge, obtained by the Röntgen-rays, of the normal development of these parts may lead to the recognition of more definite etiological facts regarding this disease and all other diseases connected with nutrition.

In rhachitis, for instance, it will be important to compare the epiphyses and the zone of proliferation at the time when a rhachitic child is examined, with these areas of a normal child of the same period of development. Many interesting questions at once occur to us—such as, Does the ossification of the epiphyses depend on the size of the child or its age, or on both?

In fact, each of us, whether physician or surgeon, or one who is making some especially careful study connected with anatomy, will find it of great advantage to enter upon his study from the standpoint of normal development.

It is with this idea that we have been making a series of pictures, which, when completed, will represent a table of reference by which we can compare abnormal conditions with the normal anatomy of the same stage of development.

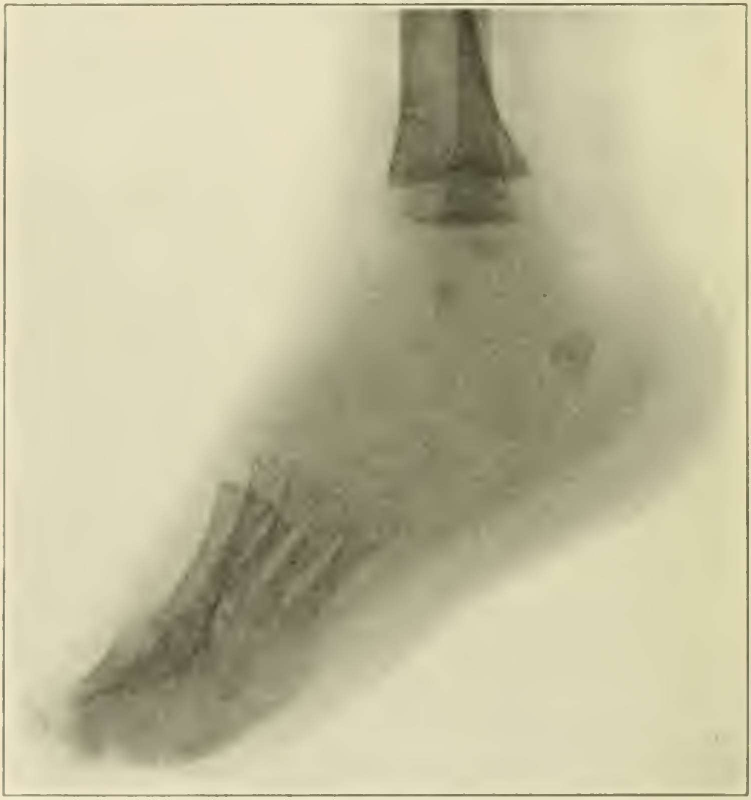

Fig. 1.—Abnormal foot of a child. (Compare with Fig. 2.)

An instance of how important to the surgeon may be this comparative anatomy in cases of anomaly is shown in Fig. 1. This is the foot of a child of three years. It shows in comparison with the normal foot of a child of three years, as shown in Fig. 2, an abnormal condition. The child was treated for some time by orthopedic surgeons, with bad results. A Röntgengram was then taken and the anomalous condition disclosed.

We shall only show in this paper a few of the series which illustrate certain interesting points and exemplify the value of such a series. The entire series will be published later. There are certain rules which must be followed if a normal anatomical progressive development is to be of practical use to the practitioner. First, it must be understood that we cannot depend on the mere age of an apparently healthy child to determine whether the picture of its organs and bones represents what should be considered the normal development at that age. Age is an unreliable standard, unless the normal development as to weight, height, the teeth, the fontanelles, and other external evidences of healthy growth are taken into consideration. It is, therefore, necessary first to make a careful physical examination of a number of children selected as representing the normal physical conditions of a certain age, and then to choose by means of the Röntgen method those pictures which show the average development at that age, and take this average as our standard.

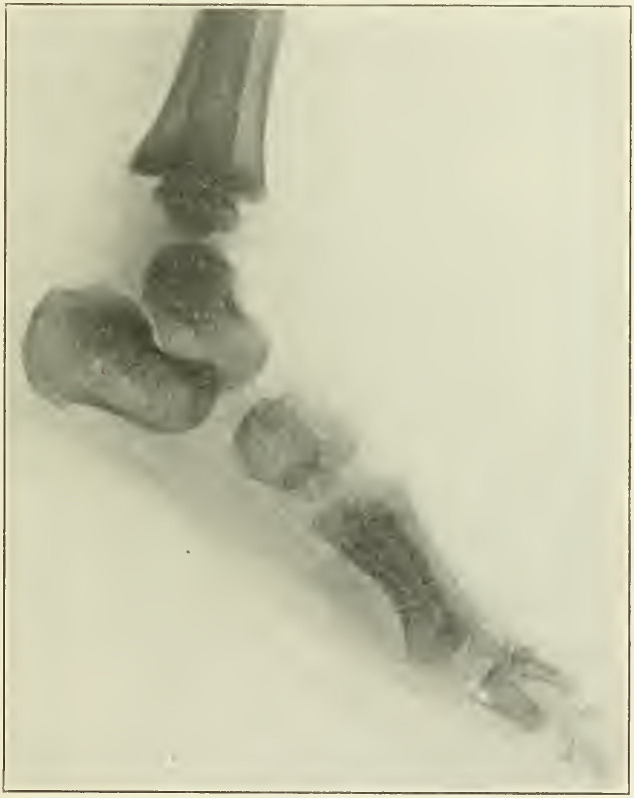

FIG. 2.—Normal foot of a child. (Compare with Fig. 1.)

There should also be a special standard technique in connection with these pictures. The tube should be in all cases at the same distance from the part taken, and the part should be in the same position in all cases. This will give us a standard of comparison, which it is very necessary to have for a critical diagnosis, and especially for exact work on operative cases. It should also be expressly stated in connection with each plate whether the picture is of actual size or reduced or enlarged, and how much. A series of this kind, extending from premature life to adolescence, will give the practitioner exact information of the most varied and important conditions of normal comparative development at a period of growth

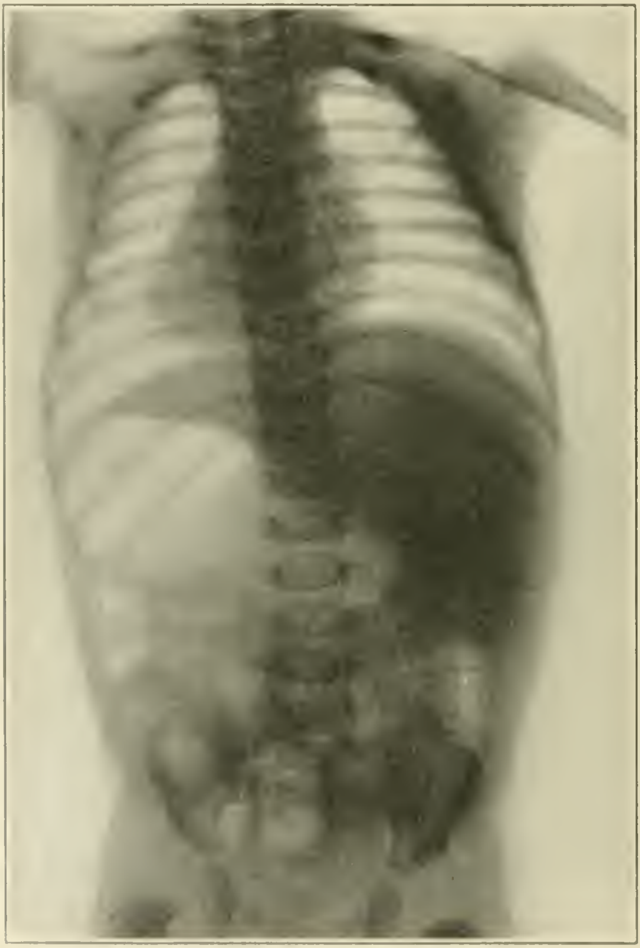

FIG. 3.—Showing the organs, especially the height of the diaphram and the cardiohepatic angle.

when such changes are most rapid, and a knowledge of them of infinite value for diagnosis and treatment.

In such a series one should note especially the shape and size of the heart, the cardiodiaphragmatic angle, so useful for a differential diagnosis between cardiac enlargement and pericardial effusion, the different centres of ossification in connection with the development of the epiphyses, the zone of proliferation, and the size and position of the stomach, liver, spleen, kidneys, and bladder.

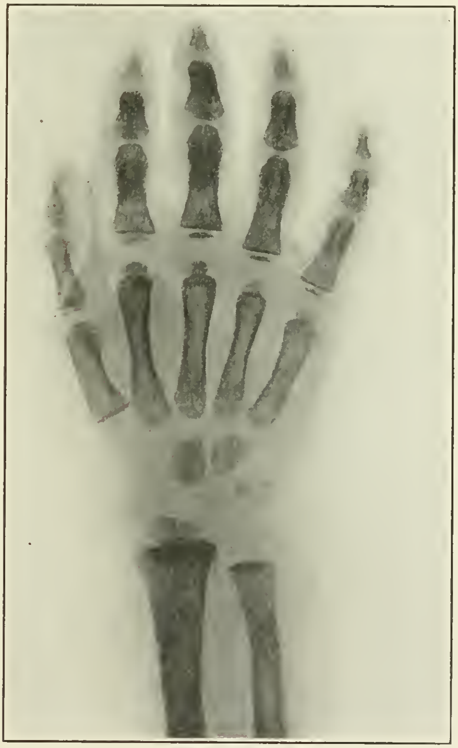

FIG. 4.—Centres of ossification in the hand at two and one-half years of age.

In looking at the Röntgengram which represents the heart in one of these pictures (Fig. 3) it will be seen that the diaphragm is quite distinct and that the recognition of its angle with the heart in a doubtful case of pericardial effusion would aid us very greatly in differentiating a normal or dilated heart from the effusion. In this series of pictures it is worth noticing that the centres of ossification are absent at first, then gradually appear as small plates, and finally assume their normal size. Figs. 4 and 5 show the changes in the centres of ossification in the hand, which take place at two and one-half years, and six years, respectively.

Finally, we would emphasize the fact that however important has been the precise knowledge of anatomical conditions studied after death, yet as supplementary to such knowledge, a knowledge of actual living conditions in a special individual is far more important and practical for the practitioner of medicine.

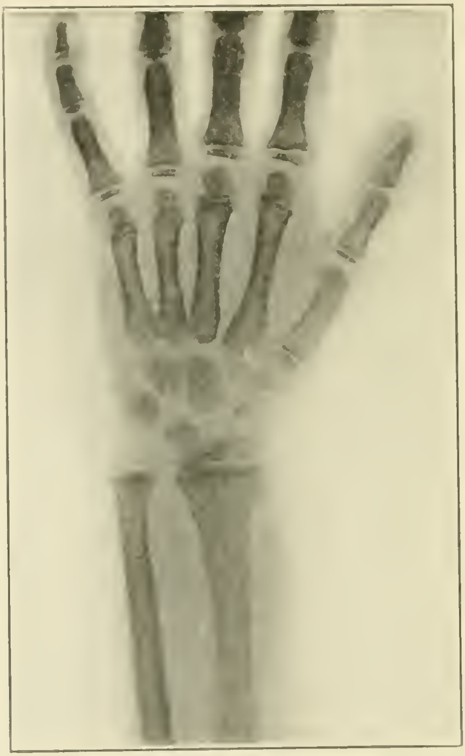

FIG. 5.—Centres of ossification in the hand at six years of age

The work upon which this paper is based was done at the Children's Hospital, Boston.

THE USE OF AMMONIUM OXALATE IN BLOOD-CULTURE TECHNIQUE.

By Albert A. Epstein, M.D.,
of New York.

(From the Pathological Laboratory of Mount Sinai Hospital, New York.)

Recent investigations in blood-culture work on special cases of infectious diseases (typhoid fever, for example) indicate clearly the necessity of making blood cultures according to more than one method.[1] The routine blood culture on a few media is a relatively easy matter; more elaborate methods cannot be employed with ease at the bedside of a patient, in either the hospital ward or private house. A complete cultural study of the blood requires a considerable outfit, the sight of which in certain cases excites fear in the patient and occasionally interferes with the asepsis necessary for the success of the culture. Apart from this minor consideration, the coagulability of the blood constitutes at times a real difficulty, and puts certain limitations on the work. It necessitates a rapid and complete distribution of the blood in the various media employed, and thus prevents its further manipulation and study. When a small quantity of blood is withdrawn from the patient (as may happen in cases with poor peripheral circulation, or in children) a uniform distribution of the blood is even more difficult, and the culture must at times be incomplete. Furthermore, the coagulability of the blood adds to the bactericidal action of its serum and is thus detrimental to the growth of bactera in positive cases.[2] It is, therefore, necessary to make the bedside procedure simple.

With this object in view, I introduced the use of a solution of ammonium oxalate, in conjunction with our routine blood-culture work, by means of which some of the blood withdrawn is kept fluid, taken to the laboratory, and studied in any manner desirable. Thus the results obtained by the latter method were controlled by those obtained in the cultures made in the usual way. The present article comprises a description of the method and a preliminary report of the work done thus far.

The solution employed in this method is made up of the following ingredients: ammonium oxalate, 2 grams; sodium chloride, 6 grams; and distilled water, 1000 c.c. The resulting solution is distributed into large test-tubes in quantities of 10 c.c. each (measured accurately by means of a pipette or measuring funnel), and sterilized on three consecutive days, twenty minutes at a time. The level of the fluid in the tubes may be indicated by some mark; this serves to control the evaporation of the fluid. The tubes are then stored in the cold until needed. It is best to make up fresh solutions every three or four weeks, as the oxalate is likely to undergo decomposition.

It is obvious from the above figures that the solution is approximately isotonic with the blood. The choice of ammonium oxalate as a decalcifying agent of the blood is based on some previous work in connection with the sterilization of blood,[3] in the course of which we found that the oxalate was satisfactory for the purpose and exhibited (in the quantity used) no apparent detrimental influences on the viability or growth of bacteria.

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[1] Libman, Johns Hopkins Hosp. Bull., July, 1906.

[2] Conradi, Münch. med. Woch., Nr. 34, 1906; Müller and Gräf, Münch. med. Woch., Nr. 2, 1906.

[3] Bernstein and Epstein, Jour. Infect. Dis., 1906.

The steps of the method are as follows:

1. About 10 c.c. of blood is withdrawn from a chosen vein, and

2. Introduced into a tube containing 10 c.c. of the sterilized ammonium-oxalate-saline solution.

3. The mixture is then shaken thoroughly; or, better still, poured into another sterile tube. The blood stays fluid. It is then taken to the laboratory and treated according to any or all of the methods suggested for blood-culture work.

The quantity of ammonium oxalate employed is really in excess of the needed amount. This is done so as to permit the use of larger amounts of blood than indicated above.

The above method has thus far been employed by us in 78 cases, taken from various patients. The results obtained with the ammonium-oxalate method have been the same as those obtained with the controls. The bacteria encountered in our positive cases were the pneumococcus, streptococcus, staphylococcus, as well as the typhoid bacillus and Bacteruim coli. The phenomena exhibited by these bacteria in our control cultures (inclusive of the typhoid-type colony)[4] were the same as those obtained in the cultures made by the oxalate blood.

The introduction of this method in blood-culture work is intended as an intermediate step, whereby the preparation and work at the bedside may be reduced to a minimum; also to facilitate more extensive study of the blood than is possible with our present methods. In addition to the ease with which blood-cultures can be taken by this method, it offers other advantages. The oxalated blood may be centrifugated, and spreads may be made from the sediment and examined microscopically for bacteria. In intense infections an early diagnosis can thus be made. The supernatant fluid obtained by centrifugalization can be employed for the determination of the opsonic index of the blood.

So far we have not had any experience with the application of this method to the attenuated organisms, such as the diplococcus and streptococcus[5] of subacute endocarditis. Our series of cases has, as yet, been too small to prove conclusively that the method may be used safely in every case. Further studies of the method, however, are being carried on in our laboratory by Dr. Ryttenberg, the results of which will appear in a publication by him.

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[4] Epstein, Proc. New York Path. Soc., Oct., 1906

[5] Libman, loc. cit.

REVIEWS.

American Practice of Surgery. Edited by Joseph D. Bryant, M.D., and Albert H. Buck, M.D., of New York City. In eight volumes. Vol. II, pp. 778, with 213 illustrations. New York: William Wood & Co., 1907.

The contents of the second volume of the *American Practice of Surgery*, which follows the first after an interval of about six months, are classified by the editors thus: Part VI, miscellaneous semi-surgical diseases, including leprosy, plague, glanders, anthrax, etc. (Leys); VII, tuberculosis (Gibney), and syphilis from a surgical standpoint (E. L. Keyes, Jr.); VIII, abscess (Jonas), ulcer (Mastin), gangrene (A. C. Wood); surgery of the skin (Douglass W. Montgomery), of muscles, tendons, bursæ, etc., (J. Clark Stewart), of nerves (Willard), and of lymphatics (Dowd); IX, burns, etc., (Tilton), and frostbite (Paul M. Pilcher); X, wounds of the soft parts (Darnell), and gunshot wounds (Borden).

It is gratifying to see the literary reputation of the medical departments of our Army and Navy so well sustained by Leys, Darnell, and Borden, in the present volume. They have proved themselves worthy successors of the lamented Lidell, of Bill, Otis, Wales, Brinton, and other military and naval surgeons of the past.

Leys discusses the various semi-surgical affections which have acquired increased importance from their frequency in the remote corners of our overgrown republic. Perhaps his remarks are a little too extended, but he writes from a full mind; and particularly to be commended are his clear, dogmatic, and complete sanitary directions for prophylaxis.

Surgical tuberculosis is treated by Gibney. He maintains the frequency of primary pulmonary lesions, and again asserts his belief that whooping-cough is a prime predisposing factor in the production of bone tuberculosis. He regards scrofula or struma as a disease due to inoculation with bovine tuberculosis, and recognizes it as a predisposing cause of bone and joint disease.

Keyes discusses syphilis in an *ex cathedra* manner, frequently substituting in undecided questions a dogmatic opinion of his own for a lucid exposition of facts from which an intelligent reader might draw his own conclusions. He gives a cheerful prognosis except for alcoholics. He condemns iodides and inunctions of mercury in the treatment of syphilis, and thinks hypodermic injections of mercury the only proper form of administration. His own preferences would probably be more cordially adopted were his ill-tempered and oft-repeated sneers at the general practitioner less prominent.

Jonas's clinical description of abscess is good; but we unreservedly condemn his treatment of *acute* abscess by curetting the abscess wall after incision. This practice is responsible for more spreading inflammations than is the original focus of infection itself. An acute abscess tends to spontaneous cure after evacuation, and even massage and kneading of the surrounding tissues are injudicious and likely to spread infection. The use of carbolic solution for moist antiseptic poultices is in our opinion of very doubtful expediency. The danger of gangrene from carbolic acid poisoning is by no means imaginary, and according to some experiences is more frequent with the feeble solution (2 per cent.) which he recommends, than it is with solutions of greater strength. We much prefer a dressing soaked in equal parts of alcohol and corrosive sublimate solution (1 to 2000), and having had extended personal experience of these two forms of treatment, can truthfully say we never saw so many infected wounds and spreading inflammations in an equal space of time, as in the service where carbolic acid was used.

The article on gangrene and gangrenous diseases fills 100 pages. It is too long; it is, however, comprehensive and exact, but it is not systematic. Repetitions are frequent, and unnecessary space is thus consumed. It is an ambitious article, but cannot satisfactorily be presented to the reader in abstract. It has apparently been the author's endeavor to cover the literature of the subject thoroughly, and to this end he seems to have abstracted nearly every article, even remotely related, which could be found in the Index Medicus. Unfortunately no discrimination is shown, those of no value (and they are many) being quoted as fully as those few from which the reader may be expected to derive some benefit. The author's personal experience with gangrene appears to have been confined to a single case; but with commendable industry, which might well be imitated by other surgeons with a dearth of clinical material, he makes it do duty, (1) as gangrene due to arteriosclerosis (p. 214); (2) as diabetic gangrene (plate xii); and (3) as emphysematous gangrene (p. 252); and all within the confines of this one article. Dr. Wood's descriptions of traumatic or emphysematous gangrene and of hospital gangrene are particularly graphic. Perhaps this is to be attributed to the fact that they are taken bodily, with little verbal change, and with no credit, from the pages of Ashhurst's *Surgery*.

Dr. Douglass W. Montgomery, of San Francisco, contributes an elaborate article, covering nearly 100 pages, on the surgery of diseases of the skin. It is a pity that anthrax, glanders, actinomycosis, etc., are included here, since they have already been treated very ably and at quite sufficient length by Leys. Among the many rare affections of the skin described, we find no mention of pilo-nidal cysts and fistulæ. The section on skin cancers is especially worthy of notice; the clinical descriptions of the course of the disease show the value of prophylaxis, and prove how inefficacious all palliative treatment eventually is; and his insistence on excision as the safest remedy might well be heeded by the hordes of practitioners and quacks employing the Röntgen rays and cancer pastes as therapeutic measures.

The concluding subjects are the most important in the volume, and the articles are in every way worthy of their titles. Too much praise can scarcely be bestowed on Major Borden's monograph on gunshot wounds. We consider it one of the most valuable contributions on the subject in the English language for the last twenty years. His article is cyclopedic, systematic, exact; his mind has a firm grasp of the subject, and he presents his ideas in an orderly and intelligible manner. He shows the difference between modern military wounds and those of civil life, the latter more nearly resembling the military wounds of the Civil War, and requiring immediate removal of the bullet, which lodges in the vast majority of cases. The wounds of modern war, however, made by the small, jacketed bullets of high velocity, seldom if ever require immediate operation, the missiles rarely lodging, and being very nearly aseptic when they do. He gives the credit for introducing this revolution in the treatment of gunshot wounds on the field of battle to von Bergman, who, as is well known, in the Russo-Turkish War of 1887, made the daring experiment of treating fifteen soldiers with gunshot wounds of the knee-joint by an occlusive dressing, instead of by immediate amputation of the thigh, which had been the recognized treatment since the days of Guthrie. The mortality from the latter treatment had always been over 50 per cent.; but only one out of von Bergman's fifteen patients died. It appears to be true, nevertheless, that the main element in bettering the ultimate prognosis in gunshot wounds is due rather to the modern missiles than to any improvement in surgical treatment; and it also seems that the few instances in which prognosis is worse now than formerly are due to the more lethal effects of modern missiles on structures where their explosive effect is apparent.

This is a large volume, and has many of the faults of the previous volume, which was recently reviewed in these columns. The articles are, with the few exceptions noted, not cyclopedic; they are ill-balanced; they overlap each other to an unwarranted degree. For these faults we think the editors are as much to blame as the authors. For the improvement in the letter press, and for the decrease of the faults mentioned, the editors are to be commended.

A. P. C. A.

DISEASES OF THE NERVOUS SYSTEM RESULTING FROM ACCIDENT AND INJURY. By PEARCE BAILEY, A.M., M.D. Pp. 600. New York and London: D. Appleton & Co., 1906.

SEVERAL years ago Dr. Bailey published a book called *Accident and Injury in their Relations to the Nervous System.* In the present work he attempts a treatise on all traumatic affections of the nervous system. If his views are to be accepted, most diseases of the nervous system may be due to trauma. This is an interesting fact and the natural query is, Why should not injuries to the chest and abdomen cause diseases of the heart, lungs, stomach, liver, and other organs? Surely if a nervous disease can be caused by a blow or an injury of some kind, should not a similar injury to the body cause disease of the internal organs? Possibly the author, when speaking of the different diseases, had in mind the literature, and not his own views. However that may be, he speaks of progressive muscular atrophy, and he includes in this disease amyotrophic lateral sclerosis, paralysis agitans, and multiple sclerosis as being due to trauma. Most neurologists will not agree with him that amyotrophic lateral sclerosis and progressive muscular atrophy are the same thing. These diseases are coming to be regarded more as the result of a lack of development of the cerebrospinal axis, an abiotrophy, the symptoms coming on in the early or in the latter part of the patient's life. Trauma may be the exciting cause of the appearance of the symptoms, but hardly the cause of the disease. The author's views that both paresis and tabes cannot be produced by trauma alone are sound, and he justly considers injuries only as a contributory cause. He favors lumbar puncture as a diagnostic measure in intermeningeal hemorrhage.

When speaking of the diagnosis of head injuries, he states that certain conditions of the pupils seem of particularly grave significance, and that he has noticed high mortality in cases with persistent and equally contracted pupils, with immobile pupils, and with pupils presenting alternating contraction and dilatation. Again, when speaking of compression of the brain, he states that among the early symptoms the pupils are contracted at first, and are unequal, the larger pupil generally being on the side of the compression; later the pupils are dilated and do not respond to light. These are the general rules he says, though subject to many exceptions. These facts are important. In the perusal of most text-books, the descriptions of the pupillary phenomena are very vague, and practically no two books agree as to what the conditions of the pupils are in injuries to the brain and skull.

The author's views on the causation of insanity as a result of injury are sound, and his treatment of injuries to the spinal cord is excellent. His conviction is against the existence of concussion of the spinal cord, but with this opinion some neurologists would be inclined to disagree.

The second part of the book is concerned with the so-called traumatic neuroses; this is the best part of the book. The descriptions of traumatic hysteria and neurasthenia are excellent, and his treatment of these subjects is good. In the last part, he takes up the medicolegal considerations and malingering. The book on the whole is excellent, a credit to the author, and should be read by everyone interested in this subject. T. H. W.

A Text-book of Pathology. By Alfred Stengel, M.D., Professor of Clinical Medicine, University of Pennsylvania; Physician to the Pennsylvania, University, and Philadelphia Hospitals. Pp. 977; 399 illustrations in the text, many of them in colors, and 7 full-page chromolithographic plates. Fifth edition. Philadelphia and London: W. B. Saunders Co., 1906.

To all those who are interested in pathology, and indeed in clinical medicine as well, this book is very familiar, and since from one edition to another the changes have doubtless been followed, it is entirely superfluous to comment upon the work in general. The fourth edition appeared in 1903. The arrangement of the present edition does not differ materially from the last edition, but very considerable change has been made in the subject matter. Many additions have been made to the part on general pathology, and in some chapters, such as the one on animal parasites, the matter has been in places recast. New matter has also been added to the chapters on retrogressive processes, inflammation, and regeneration, and to that part of Chapter VII which deals with the problems of immunity. In Part II, which is devoted to special pathology, changes and additions are also noticeable. With these alterations and the new illustrations, the book has been enlarged about fifty pages. It remains as it always has been, a most useful book. W. T. L.

Diagnosis of Organic Nervous Diseases. By Christian A. Herter, M.D. Revised and enlarged by L. Pierce Clark, M.D. New York and London: The Knickerbocker Press, G. P. Putnam's Sons, 1907.

This little book, of nearly 700 pages, is the revised edition of the earlier work of Dr. Herter; Dr. Clark revised the present edition. The scope of the book has been somewhat enlarged and its title has been changed to the present one. It is an excellent book for the student of medicine. The arrangement of the text is different from that in other compends, in the fact that the different subjects are treated according to symptomatology.

The diagnosis of the different nervous diseases is constantly kept in the foreground, and little space is devoted to treatment. It is difficult to criticise the subject-matter in a book like this, in which only a bare outline is given of symptoms and principles. It seems, however, that there is too much repetition of old theories and methods. For instance, the motor centres have been placed both in front and back of the central fissure, and it is presumed from the text that the sensory functions are localized in the same areas.

Again, when discussing the effects of lesions of the inferior parietal lobe, ptosis is mentioned as a probable symptom. No mention is made of stereognostic function as being localized in the parietal lobule. When speaking of the tumors of the brain, the authors state that syphilitic and tuberculous intracranial growths are more common than other varieties. This does not agree with the experience of other neurologists who believe that sarcomas are the most common. On the whole, the book is a good one, and deserves the reputation it has made for itself. T. H. W.

TUMORS, INNOCENT AND MALIGNANT: THEIR CLINICAL CHARACTERISTICS AND APPROPRIATE TREATMENT. By J. BLAND SUTTON, F.R.C.S., Surgeon to and Member of the Cancer Investigating Committee of the Middlesex Hospital, London. Fourth edition; 355 engravings. Chicago: W. T. Keener & Co., 1907.

WITH the first publication of this book its usefulness was immediately recognized. One edition followed another in rapid succession, and now in the fourth we find many valuable additions. This new matter includes a short account of the prevailing theories as to the origin of cancer, additions to the section upon chorion epithelioma, chapters upon tumors of the ovary and testes, and a chapter upon heterotopic teeth. Of these additions perhaps those of most interest generally are the chapters upon tumors of the ovaries and testes. These chapters are excellent. As regards the cause of cancer the author makes no definite statements. Three hypotheses are discussed—the embryonic, the parasitic, and the biological—but the author's view of the whole question is summed up well in the last sentence of this chapter when he states that "whilst investigators are hunting for the cause of malignant tumors practical surgeons have to deal with the concrete disease." Indeed, this sentence may be taken to typify the book, for it is essentially practical and of real use to the surgeon. Minor alterations have been made throughout the volume and a number of new illustrations have been added.

We still find the chapters upon cancer of the breast, a subject of enormous interest and practical importance to the surgeon, strikingly inadequate. The chapters upon tumors of the thyroid gland is also very disappointing, but in spite of a few shortcomings it is evident that the book, good at the start, improves with each edition.

W. T. L.

A MANUAL OF PATHOLOGY. By GUTHRIE MCCONNEL, M.D., Pathologist to the St. Louis Skin and Cancer Hospital, and to St. Luke's Hospital, St. Louis, Mo. Pp. 523. Philadelphia and London: W. B. Saunders Co., 1906.

THE author's purpose in writing this manual was to condense into a small book for the student important points which receive a broader treatment in most text-books upon the subject. From a book such as this, it is, of course, not possible to study pathology, but for a rapid review of the subject it may be useful. The arrangement of the book is much the same as is adhered to in most of the larger text-books: Part I deals with general pathology; Part II is devoted to special pathology. In the last three chapters of Part I, postmortem technique, laboratory technique, and bacteriological methods are briefly discussed. It is naturally difficult in a book of this sort to include the results of recent investigations, and the author cannot digress from the older classifications of diseased processes; still, with our present knowledge it seems out of place to class pernicious anemia, leukemia, and Hodgkin's disease as diseases of the blood, and to designate the spleen as a ductless gland. Quite a number of illustrations reproduced from the larger books of Durck and McFarland are scattered through the volume. W. T. L.

A TEXT-BOOK OF PSYCHIATRY. A PSYCHOLOGICAL STUDY OF INSANITY FOR PRACTITIONERS AND STUDENTS. BY E. MENDEL, M.D., Professor in the University of Berlin. Authorized translation, edited and enlarged by WILLIAM C. KRAUSS, M.D. Pp. 311. Philadelphia: F. A. Davis Co., 1907.

THIS compend was written for the practitioner and the student in Germany, and has been translated for the use of the American student and practitioner. There is no subject in the entire realm of medicine more difficult of presentation than that of diseases of the mind. The science of psychiatry up to the present time has been essentially a science of classification of the symptom groups and so-called diseases. In the absence of a distinct pathology any distinctive group of symptoms can be considered as a clinical entity. Each author, according to his experience, presents the subject in a simple or more or less complex fashion. As a result of this, it is practically impossible for the reader to get a clear idea of the subject of mental diseases from a text-book, without extensive clinical experience. The specialty as taught to the student leaves him, as a rule, at the time of graduation, with a very confused idea of diseases of the mind. In order to present the subject in such a way that it comes within the grasp of the mind of the student or practitioner of medicine, the author must have an extensive experience with mental diseases. From such a standpoint, no one is better qualified for such a task than Mendel. His very extensive clinical and teaching experience has enabled him to present this subject in a clear, concise, and systematic manner.

In the absence of a distinct pathology, the subject-matter is presented upon a psychological basis. In other words, the book presents a psychological study of insanity. The first half of the book is devoted to a very careful consideration of the symptomatology of insanity. This is divided into a consideration of the disturbance of sensation, sense perception, memory, feelings, emotions, consciousness, actions, speech, and viscera. The contrast between the actions of the normal and the pathological mental states is very clearly shown and gives the student a peg upon which to hang the isolated descriptions, etc. The English at times, is somewhat obscure, but this is to be expected in dealing with psychological states. The term "inheritor," as used to designate those who show neurotic ancestry, does not clearly express what is meant. In other respects, this portion of the book is worthy of the highest commendation. The second half of the book is devoted to special psychiatry. The classification used is as simple as possible for working purposes and at the same time takes into consideration the more recent advances in the subject. The systematic presentation of the different types of insanity, the clear-cut descriptions of the different symptoms, the psychological analysis of the disease as a whole, and the elimination of unnecessary discussion, gives this portion of the book the stamp of a well-ordered mind and one accustomed to the difficulty of teaching insanity. The author's attitude toward such subjects as moral insanity is tinged by his long experience with the forensic side of medical jurisprudence. Not only in connection with the subject of moral insanity, but elsewhere throughout the work, his method of looking at the subject from both the medical and legal standpoint, gives an added value and logical simplicity to the descriptions and analyses. More attention than usual is paid to the subject of hallucinatory delirium. Twilight mental states are also very carefully considered. A short chapter is devoted to the arteriosclerotic psychosis.

It is impossible to imagine more subject matter than is here presented, contained in such a small volume without a loss of clearness of description. It should be recognized by an author of a textbook of insanity that a very extensive work with a full consideration of the many diverse views will neither be read nor understood by the student or practitioner. The reviewer of this book has kept the standpoint of the student in view. The expert in mental troubles will, however, find many things of value, and especially the clear and systematic presentation of a difficult subject.

In the translation, the German text is closely adhered to without, however, any sacrifice of the natural, clear, and direct style. The few illustrations could well be improved upon. Apart from this, the book is well printed in good type and well bound.

In conclusion, particular attention should be directed toward the classification of the different types of insanity and the table of contents. Were it possible to have institutions for the insane adopt this classification, much value from a statistical standpoint could be gained from their annual reports. D. J. McC.

A Handbook of Skin Diseases and their Treatment. By Arthur Whitfield, M.D. Lond., F.R.C.P., Professor of Dermatology in King's College; Physician to the Skin Department, King's College and the Great Northern Central Hospitals. Pp. 320. New York: Longmans, Green & Co. London: Edward Arnold, 1907.

As the title plainly indicates, this is a handbook and not a treatise. The general arrangement of the subject-matter, and indeed the handling of the matter itself, is different from that of the usual small text-book. The contents and the manner of dealing with the entire subject call to mind a similar-sized volume by Pye-Smith, of London, published some years ago, and which was deservedly well received by the profession. Dr. Whitfield has written for the purpose of giving the information he has to convey from his own point of view, and without regard to the traditional style of handbooks. Everything is brief, terse, and to the point. No classification is attempted: this is to be regretted.

Under the head of general therapeutics, it is stated, "apart from the remedies for various well-defined constitutional errors, only three drugs need be considered, namely, arsenic, mercury, and thyroid extract." From this it is obvious that the author is most conservative in the use of drugs. We quite agree with the remark that mercury is often of value in nutritive disorders of unknown nature, and that it is by no means safe to conclude that a skin disease is syphilitic because it is found to yield to internal mercurial treatment. The chapter on pyogenic infections, together with a description of A. E. Wright's method of treatment, to which the author has experimentally given attention, is both original and interesting, brief as it is.

The "ringworms," so common in London, are elucidated according to recent studies, and Sabouraud's teachings largely followed. The Röntgen rays, it is stated, merely remove the hair, but do not cause the death of the fungus. Pityriasis alba, occurring in the scalp in scaly patches encircling the hair, the hair itself escaping invasion, is considered to be due to the "bottle bacillus." This affection the author states is fairly common in England, but in this country it is not generally recognized. Our best recent treatises do not describe it. There is a chapter on "tumors," but the lesion is not defined; some epidermic formations are included here. Ichthyosis, leukoderma, and scleroderma are grouped together in one chapter. The term "syphilide" is employed – rather the better English word syphiloderm, the latter being for every reason preferable.

Sound advice is given for hypertrichosis, which if followed would in the end save the patient both suffering and disfigurement. Depilatories are condemned, the safety-razor being preferable. The Röntgen rays should always be employed with caution.

While there is much in many directions that is original and interesting in the book, therapeutics cannot be designated its strongest feature. Not much is said in general on the internal administration of drugs, nor is the matter of diet strongly insisted upon in diseases in which a dietary really often plays a prominant part in the cure.

There are some good photographic illustrations and photomicrographs of sections, but no attempt is made to illustrate the diseases fully. The style of the author is easy and unaffected, so that one may peruse any page with pleasure as well as profit. L. A. D.

NOTES ON BLOOD-SERUM THERAPY, PREVENTATIVE INOCULATION, AND TOXIN AND SERUM DIAGNOSIS FOR VETERINARY PRACTITIONERS AND STUDENTS. BY WALTER JOWETT, F.R.C.V.S., D.V.H., Formerly Demonstrator of Comparative Pathology and Bacteriology in the University of Liverpool. Pp. 200. Chicago: W. T. Keener & Co., 1907.

DR. JOWETT in this little book endeavors to give a short account of the subject of immunity and the vaccines, sera, and toxins used in present-day veterinary practice for the prevention, treatment, and diagnosis of disease. The first chapter deals with the principles of immunity in general. Naturally the matter is condensed into a small space, but even this mere outline of the subject is satisfactory and sufficiently clear to convey the main underlying principle to the reader. In the second chapter the methods of conferring immunity are reviewed. To each specific disease, such as fowl cholera, anthrax, swine plague, and tuberculosis, one or several paragraphs are devoted. The method of conferring immunity in such diseases as rabies, variola, etc., diseases which the author terms as those due to "ultravisible viruses," are described in Chapter III, while the same system is used, regarding diseases caused by protozoa, in Chapter IV. Finally, in Chapter V toxins and serum diagnosis are considered. The book should be useful to anyone who is interested in comparative medicine and should certainly be read by all practising veterinarians.

W. T. L.

The Muscles of the Eye. By Lucien Howe, M.A., M.D., Professor of Ophthalmology, University of Buffalo, Buffalo, New York. In two volumes. Vol. I, Anatomy and Physiology, including Instruments for Testing and Methods of Measurement. Illustrated. New York and London: The Knickerbocker Press, G. P. Putnam's Sons, 1907.

This is the first of two large volumes the subject-matter of which is the muscles of the eye. It is undoubtedly the most extensive work in the English language upon the special subject with which it deals. The writer concerns himself equally with the scientific and the practical; from consideration of the normal, the abnormal is deduced by easy and logical steps. He does not simply aim at giving just as little knowledge as is necessary to enable the reader painfully to pick his way through the manifestations of such morbid phenomena as come within the scope of the practising oculist, but he takes up the subject from the scientific stand point very much as a text-book upon one of the physical sciences deals with its subject. Works having the scope of this one are rare in any department of medicine, while those planned upon the ordinary medical text-book method are put forth in abundance. Books like this require profound acquaintance with the literature of the subject which has to be sought in the special publications of the civilized world.

The author tells us in his preface that almost the entire work was submitted to a student who had received hardly more than a high-school education. Any passage not entirely clear to this critic was rewritten or stricken out. This method should certainly render any writing to which it is subjected clear and easy of comprehension. The book deserves, and will no doubt find, a place in the hands of all who are interested in scientific and practical ocular myology.

T. B. S.

A Treatise on the Motor Apparatus of the Eyes. Embracing an Exposition of the Anomalies of the Ocular Adjustments and their Treatment, with the Anatomy and Physiology of the Muscles and their Accessories. By George T. Stevens, M.D., Ph.D. Illustrated with 184 engravings, some in colors. Pp. 496. Philadelphia: F. A. Davis, Co., 1906.

The appreciation of this work is greatly facilitated by considering it separately from the standpoint of anatomy and physiology on the one hand, and clinical and pathological positions on the other. The anatomical and physiological portions, which occupy a considerable part, offer a clear and comprehensive account of the oculomotor apparatus; the clinical portion opens a wide field which has given room for extensive controversy.

The present book is a sequel to the author's work on *Functional Nervous Diseases* published in 1884. That work was of moment in American ophthalmology in emphasizing the role of the external ocular muscles, supposed to be of prime importance in the causation of many obscure nervous affections. The controversies which some of the claims therein made excited and the opposite opinions as to their validity are well remembered. Certain contentions of the author in that work were exaggerations and did not stand the test of time and experience; and although he declares that the present work is intended to promote the development of the central doctrine of the work just mentioned, it departs very considerably both in its claims and conclusions from the earlier work, and, as we believe, decidedly for the better. T. B. S.

Essentials of Obstetrics. By Charles Jewett, A.M., M.D., Sc.D., Professor of Obstetrics and Gynecology in the Long Island College Hospital; assisted by Harold F. Jewett, M.D. Third edition, revised and enlarged. New York and Philadelphia: Lea Brothers & Co., 1907.

This little book serves a useful purpose in enabling the student to distinguish the important portions of the subject under consideration. It is in no sense a quiz compend, but is just what its title indicates—an epitome of obstetrics. It should not be used as a crutch; indeed the, author definitely warns against this—a warning which if taken to heart by the student completely obviates the danger always connected with books of this type. The subject-matter is well and thoroughly covered; a number of additions have been made to this the third edition to keep it abreast of the times. W. R. N.

PROGRESS
OF
MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF
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A Study of Latent and Recurrent Malarial Infections and the Significance of Intracorpuscular Conjugation in the Malarial Plasmodia.—CRAIG (*Jour. Infect. Dis.*, 1907, iv, 108) gives a most thorough analysis of latent and recurrent malarial infections; by the first (latent), meaning those cases in which the plasmodia may be demonstrated in the blood, but in which no clinical symptoms of sufficient gravity to attract attention are to be observed, and in the latter (recurrent), the appearance of symptoms due to the same group of parasites that caused the original infection. The importance of the latent type is great and amounted to nearly 24 per cent. of the cases examined, and as these cases showed no clinical symptoms and were discovered only in the routine examination, these men might have been sources of infection for other persons for weeks or months. These men were mainly soldiers returning from the Philippines. The latent infections were often associated with other diseases and their importance in treatment was very great aside from the general treatment of the associated diseases. As regards the recurrent infections Craig concludes that in the estivo-autumnal tertian infections relapses occur most frequently between the twentieth and fortieth days after the initial attacks, and in benign tertian infections between the fifteenth and twenty-second day. In the etiology of these conditions Craig believes that intracorpuscular conjugation is very important and plays a great part; it is noted especially in the latter part of the acute attacks and is probably the process intended to maintain the malarial infection in the blood of man; it occurs whenever the races of plasmodia are in danger of dying out from repeated sporulation in the usual manner.

His conclusions are: (1) Intracorpuscular conjugation is the chief cause of the maintenance of malarial infection. (2) It maintains malarial infection by producing a resting or zygote stage of the plasmodia within the human body which is resistant to quinine and other injurious influences. (3) It is the cause of latency and recurrence of malarial infection, the zygote stage remaining dormant or latent until conditions are favorable when it gives birth to several young plasmodia, thus causing a recurrence of the infection. These conclusions are justified by the following considerations: (1) The presence of the process in all acute and recurrent infections. (2) The fact that during the time consumed in conjugation in all other protozoa, provided the conjugation is asexual, many generations of the organism could have been produced by division or sporulation in the usual manner. (3) The fact that such a resting or zygote stage must exist as proved by the recurrence of the infection after the discontinuance of quinine given for long periods of time. (4) The fact that in cases that have been treated at once with sufficient doses of quinine for a sufficiently long period, intracorpuscular conjugation is never seen and in such cases relapses are very rare, if they ever occur. (5) The presence in cases in which the process is most marked, of numerous large, pigmented bodies in the blood both intracellular and extracellular, which are not seen in cases in which the process is absent. (6) The argument from "analogy," which indicates that the significance of the process of asexual conjugation in the malarial plasmodia is similar to the same process in other of the protozoa.

The Influence of Subcutaneous Injection of Liver Extract upon the Hepatic Metabolism of Uric Acid.—L. B. STOOKEY (*Jour. Med. Research*, 1906, xv, 321) in association with A. S. GRANGER has previously shown that the subcutaneous injection of liver extract (dog) may lead in the same animal to an increased elaboration of nitrogenous end products into urea, and that liver extracts heated to 55° C. were not found to have this stimulating action. Attention was, therefore, directed to the possibility of an enzymotic formation of urea. These same studies were directed upon the hepatic metabolism of uric acid. Stookey reviews the literature bearing on the destruction of uric acid by the animal organism, especially as regards the liver and other organs, and the dependence of destruction on the activity of an enzyme which has been called a "uricolytic ferment." Stookey made extracts from the liver of several dogs, then made daily injections of amounts varying from 10 to 50 c.c., and, at the conclusion of each experiment, the uricolytic power of the liver was determined by hashing the organ finely and estimating the amount of uric acid destroyed in a certain time. Control experiments were made on normal dogs. The results showed a noticeable increase in the uricolytic power of the liver of treated dogs over that of the normal animals.

The Electrical Conductivity of Blood and Urine in Health and in Disease and as a Test of the Functional Efficiency of the Kidney.—DAWSON TURNER (*Edin. Med. Jour.*, 1907, xxi, 318) has been experimenting for some time on the electrical resistance of various kinds of urine in states of health and of disease, and from about 1000 observations, has determined that the observed resistance of normal urine amounts to about 250 ohms in his apparatus. To avoid polarization the estimates were made by means of a wheatstone bridge, with alternating currents, and a telephone, and at a temperature of 65°F. In artificial urines it was found that the resistance depends only upon the ionic concentration (salts, acids, and bases), and of these NaCl is by far the most important. The resistance for practical clinical purposes can be regarded as being unaffected by the presence of albumin, sugar, blood, and other non-electrolytes. Turner's tables show these facts very well. In health the resistance is low and the lower it is, other things being equal, the healthier the condition. In certain acute and chronic diseases a very high resistance is found constantly present. In pneumonia, diabetes mellitus (increased resistance possibly depending somewhat on the increased viscosity), kidney diseases, and pernicious anemia, the resistance is high. Turner also found it double the normal during sick headaches.

As regards the blood the average observed resistance in normal persons was about 700 to 900. The resistance depends upon a salt concentration of the blood and the relative volume of the corpuscles to the serum. The greater the salt concentration the lower the resistance, but the greater the number of the red cells present, the higher the resistance. Hence a high resistance of the blood and a low resistance of the urine indicates health. The resistance of the blood in kidney diseases, chlorosis, anemia, and pernicious anemia is low and in the latter may be half that of normal blood. Comparing systematically the resistance of the blood and urine Turner has established a ratio called the "hemorenal salt index," or the ratio of the electrical resistance of the blood to the electrical resistance of the urine. "In health this would be equal to 3, 4, or 5 whole numbers, namely, $\frac{900}{225}=4$. The higher this number the healthier, other things being equal, is the condition of the patient."

If the index increases, it indicates that the blood contains fewer salts or is richer in corpuscles, that the urine contains more salts, and that the functional activity of the kidney is increasing; if the renal index diminishes, the kidney is becoming sluggish and inefficient.

Turner reports upon several diseases and shows an interesting table of 19 different patients. In anemia the blood corpuscles can be allowed for by count and the electrical resistance depends then solely on the nature and amount of salts present. A low hemorenal index will then mean that the blood contains too great a proportion of salts and the urine too small a proportion, or renal inefficiency. The kidney instead of withdrawing salts from the blood and secreting them in the urine is allowing them to be retained in the blood. Turner looks upon an index below 3 as abnormal and below 2 as serious. Surgical interference in kidney lesions is contra-indicated if the index is much below 3. More comparisons of this method with cryoscopy are needed to determine their relative values which have already shown in some instances an agreement. At any rate this is a much more rapid method of clinical diagnosis than cryoscopy while only one drop of blood (5 c.mm.) is required instead of 10 to 20 c.c. for cryoscopy. This determination of the hemorenal salt index is another example of the application of precise scientific measurement to clinical medicine and surgery.

Calcification of the Breast following a Typhoid Abscess.—W. S. Thayer and H. H. Hazen (*Jour. Exp. Med.*, 1907, ix, 1) report the case of a patient with typhoid fever and intestinal hemorrhage who had taken, during eleven days, 132 grams of calcium lactate by the mouth as well as 5 grams of calcium chloride subcutaneously. Eleven days after the subcutaneous injection there developed a large abscess of the breast from which Bacillus typhosus and Staphylococcus aureus were obtained on culuture. The abscess was opened and the wound packed with iodoform gauze. Eleven days later a deposit of calcium appeared in the granulation tissue of the wound. This rapidly increased until the rim of the opening and the lining granulation tissue were converted into a hard, calcareous mass. A number of nodules of an apparently similar character appeared in other parts of the breast. A month later, the cavity showing slow improvement, the packing was removed and a simple, plain, protective dressing was used. On the basis of the experience of Gerhardt and Schlesinger, who found that under a carbohydrate-free diet the calcium excretion was increased, the patient was put on a carbohydrate-free diet. Improvement was immediate, the wound gradually healing with the complete disappearance of the calcareous deposits. The patient slowly gained weight and left the hospital in excellent condition.

Attempts to reproduce the picture in rabbits failed. Studies were also made of the intake and output of calcium. On a diet of milk and eggs the patient showed a material calcium retention; during the carbohydrate-free diet there was a considerable excess of calcium elimination over the intake. On a normal ward diet there was a slight retention of calcium. The percentage of calcium eliminated in the urine as compared with that eliminated in the feces was much higher during the periods of carbohydrate-free diet.

With regard to the cause of the calcification Thayer and Hazen say: "We cannot speak with certainty. The remarkable association of the deposition of calcium at the seat of an injection of calcium chloride in an individual who had been receiving large quantities of lactate of calcium by mouth—a deposition occurring in tissues which were in direct connection with or in the immediate neighborhood of a dressing containing iodoform, a poison which in certain animals produces degenerative changes in the kidneys and liver which are peculiarly prone to calcification, is striking and worthy of note.

"In how far the decalcification of the affected area may have been due to the carbohydrate-free regime, with the consequent excessive calcium elimination, it is impossible, on the basis of a single case, to say. The coincidence in time of the disappearance of calcium from the breast with this treatment is, however, remarkable and extremely suggestive."

Anomalies of Respiration in Young Soldiers, Especially as Regard the Apices of the Lungs.—G. H. Lemoine (*La presse médicale*, 1907, x, 73) has been examining men for enlistment in the army and has followed closely later those who showed some slight anomalies of respiration. In most cases excellent results have followed the regular life in camp or barracks which, in some respects, approaches that in a sanitarium. Of 945 men examined, 99 showed some change in the auscultatory sounds. In the first group a change in inspiration was noted under the right clavicle in 64 men, about 6.7 per cent., all of these showing a fixed and persistent diminution in the intensity of inspiration;

14 others showed, in addition, a slight prolongation of expiration. In about 18 of these men there had been a previous pleurisy, and in many there was a family history, of tuberculosis. Their general condition and fitness for work were carefully considered before admitting them for duty, while they were afterward carefully watched and, as soon as untoward signs were manifest, were immediately dismissed for treatment elsewhere. In most instances the men picked up quickly under the carefully regulated life and soon threw off their respiratory changes.

In the second group there were 9 men with some changes on inspiration under the left clavicle, where the inspiration was rough and low-pitched, and at times associated with some prolongation of expiration. Most of these men also improved while in the army. Aside from those men with chronic pleurisy, one showed a "respiratory insufficiency" of the whole lung. He gained steadily in weight and left the service in splendid health.

Thus, of the 99 men showing some pulmonary changes, only 19 had to be definitely dismissed, and all of the others did very well. Lemoine, in his many examinations, has noted that inspiration is stronger on the right in right-handed persons and vice versa for left-handed persons, and that the more the right arm is used the more marked this difference. He asserts that especial attention should be paid to the family history only in those cases with some slight anomalies of respiration, as in 79 men with a family history of tuberculosis, although showing no respiratory changes, only *one* developed tuberculosis, whereas in the 99 here examined, about 19 had to be dismissed.

Examinations should be made three times at a month's interval, and special stress should be laid on the good general healthy condition while in service. On the first appearance of any slight weakness in work or fatigue on exertion, but without any change in the lungs themselves, the men should be immediately dismissed for more careful treatment and supervision elsewhere; but the army life is apparently more hygienic and healthful to most of this class of cases than their crowded life at home, or at work.

Interauricular Insufficiency.—H. Roger (*La presse médicale*, 1907, xi, 85) asserts that there is a certain number of cases in which all the symptoms, such as cyanosis, dyspnœa, etc., point to valvular heart disease, while a most careful examination reveals nothing definite in the heart or lungs, and reports 2 such cases which he classes as "interauricular insufficiency." The first of these was in a man, aged forty-four years, strong and apparently in good condition. His previous history up to the age of thirty years was negative. At that time he suffered from some acute pulmonary trouble which caused much dyspnœa on slight exertion. Every winter since he had recurring attacks of bronchitis, inducing rapid asphyxia and cyanosis. On admission the patient showed intense dyspnœa and cyanosis and was somewhat asphyxiated. Venesection and other measures were of temporary benefit, but the patient finally succumbed three days after admission. Examination showed severe bronchitis and emphysema of the lungs, and the heart was apparently negative, but on account of the intense cyanosis and the lack of evidence of valvular disease, the diagnosis of a communication between the auricles was made, which was confirmed at autopsy.

In the other case a man, aged fifty-four, was under treatment for a bronchitis associated with an old emphysema. The intensity of the cyanosis and dyspnœa, with an apparent absence of valvular disease, led to the same diagnosis as in the first case—"interauricular insufficiency" The cyanosis quickly reappeared on any violent exertion, especially when coughing. The patient left the hospital in a much better condition, so that there was no anatomical proof of the correctness of the diagnosis.

As regards the cause of these symptoms, Roger found that in his first case the heart, though normal in appearance, allowed water to flow from the right auricle to the left auricle through the valve, but not in the reverse way. And this same fact has been noted constantly when carefully searched for at autopsy, even though there be no apparent connection between the auricles. Thus, the intense cyanosis and dyspnœa could be accounted for by the large mixture of venous and arterial blood, due to the increased pressure in the right auricle as compared to the left, this pressure being caused by the various pulmonary conditions or severe coughing spells. Roger noted no auscultatory abnormalities in the heart in these 2 cases, but says that at times a late diastolic or systolic double murmur may be heard to the left of the sternum. Treatment is usually of only transient benefit and after each attack the recovery is slower and less complete. Roger regards this condition as an insufficiency of the interauricular valve or more simply "interauricular insufficiency."

SURGERY.

UNDER THE CHARGE OF

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ASSISTANT SURGEON TO THE UNIVERSITY AND THE PHILADELPHIA GENERAL HOSPITALS, AND INSTRUCTOR IN SURGERY IN THE UNIVERSITY OF PENNSYLVANIA.

Primary Carcinoma of the Appendix.—ZAAIJER (*Beit. z. klin. Chir.*, 1907, liv, 239) says that in 2232 cases operated on for appendicitis 18 of the appendices were found to be carcinomatous. He thinks that this number is much too low, since of the 2232 cases only a small part were systematically examined, and by such an examination undoubtedly others like his own two, macroscopically unsuspected, would have been found. He agrees fully with Baldauf that carcinomas of the appendix represent about 1 per cent. of all cases of appendicitis. It is probable that chronic inflammation of the appendix plays an important part in the development of cancer in this organ. Zaaijer says that 33 cases of appendix carcinoma have been found by operation and only 14 by postmortems. Undoubtedly the greater number of operations for appendicitis, if not the cases of appendicitis itself, occur in the second, third, and fourth decades; so that the frequency of appendix carcinoma is that of this period. Since cancer of the intestine is most frequent in the fifth and sixth decades, we see here support of the view that appendicitis is of etiological importance to carcinoma of the appendix. Zaaijer thinks that the very small cancers of the appendix require from ten to thirty years to become the large, fully developed tumors of the intestine, that is, those giving distinct clinical symptoms. In other words, a cancer of the appendix probably requires about ten years to become a recognized cancer of the cecum. Histologically, so far as they have been investigated, these appendix carcinomas have a very malignant character. The histories of the patients show that the symptoms have been those of a simple appendicitis, usually of a chronic recurrent type, and that their carcinomatous nature has not been recognized until the operation. The fact that about 1 per cent. of all appendicitis cases show carcinoma is an additional reason for operation in appendicitis. When a tumor is found at operation it will probably be a cancer. In doubtful cases a piece should be removed, and if possible examined by frozen section. If the findings are positive, the mass, the mesentery, and the associated lymph glands should be thoroughly removed.

Concerning the Methods of Operation in Obturator Herniæ.—BORSZEKY (*Beit. z. klin. Chir.*, liv, 1907, 350) says that of the rarer herniæ the obturator is the most difficult of diagnosis. Even when it is incarcerated, only now and then is the diagnosis made. In the presence of symptoms of intestinal obstruction we usually examine the usual hernial orifices, with which we are satisfied. As a rule there is no considerable swelling, so that only by a very careful examination could we recognize it. The external signs depend upon whether it passes completely or incompletely the obturator canal. If it is complete a swelling may appear immediately below the os pubis to the inner side of the femoral vessels, above the obturator internus muscle and covered anteriorly by the pectineus. When it does not pass completely through the canal, it either forces its way between the upper and middle fibers of the obturator externus or between the two layers of the obturator membrane, then remaining behind the obturator externus. It is then covered by two muscles, the pectineus and obturator externus. The thigh should be flexed, adducted, and rotated somewhat outward. If it is not palpable pressure about the junction of the lower and middle thirds of Scarpa's triangle, producing pain, should arouse suspicion of this hernia, especially if it is increased by abduction and internal rotation of the thigh. Rectal or vaginal examination may be of value, as may pain along the obturator nerve. This symptom is not found in all cases, not even in the incarcerated. Borszeky found it mentioned in only about one-third of the reported cases. With a narrow canal or with a sac full of fluid, it will probably not be absent. It loses much of its diagnostic value from the fact that it is caused by other conditions, as the pressure of a perimetritic exudate (Sachs), or a mass in the iliac fossa (Krönlein). In a large number the hernia was discovered at autopsy. Usually incarceration first compels the patient to seek relief. As soon as the diagnosis is made we must choose between reduction by taxis and operation.

The conditions are not favorable for the former; since the hernia cannot be grasped or palpated on account of the overlying muscle, while the canal is so narrow that violent pressure could easily be dangerous. Taxis by the vaginal route, according to Lejars, is also dangerous. The most important question, therefore, is: By which path can we most safely approach the hernia for operation? The external operation has many dangers because of the depth and obscurity of the field of operation and the proximity of the large bloodvessels. Lejars recommends the lithotomy position and an incision from the inner side of the femoral vessels to the tuberosity of the ischium. When the sac is opened the canal is widened and the bowel reduced. If it is gangrenous it is resected, a very difficult operation. The danger of hemorrhage from increasing the size of the canal, due to the involvement of the closely associated obturator vessels, is considerable, but the greatest danger arises from handling the gangrenous bowel by the external operation. It may rupture and the contents invade the peritoneal cavity. The operation done from the inside is much better, and in some cases the operation begun from the outside must be ended as a laparotomy. In some cases the diagnosis of internal obstruction is made, and the laparotomy done to correct it serves for the reduction and further treatment of the obturator hernia.

Rupture of the Lung Without Fracture of a Rib.—Schwartz and Dreyfus (*Revue de chir.*, 1907, xxvii, 765) report a case in which there was contusion of the lung without fracture of a rib. There was no considerable laceration of the lung, but at autopsy there was found evidence of multiple small hemorrhages. This condition explained the intense respiratory phenomena, coincident with the insignificant hemorrhage and comparatively little intrapleural hemorrhage. Schwartz and Dreyfus collected 36 cases of this kind, although they were frequently associated with multiple traumatisms, as fractures of the skull or extremities. While no fracture was discovered in Schwartz and Dreyfus' case, there was found a chondrosternal dislocation of the first two right cartilages. This and the marked ossification of the thoracic cage caused a violent compression of the corresponding part of the lung. The evolution of these small hemorrhages is often, unfortunately, the same as in Schwartz and Dreyfus' case. The contused area becomes a focus of infection with resulting traumatic pneumonia, which carries off the patient.

The prognosis of contusions of the lung is always grave, although in the 29 cases collected by Diouis du Sejour there was 60 per cent. of cures. Of the 7 cases added to this list by Sewartz and Dreyfus 5 died and 1 of the 2 that were cured was doubtful. Death occurs from asphyxia, hemorrhage, or a later complication, as infection. Very often this infection causes a traumatic pneumonia. The symptoms may be very mild and in such a case must be looked for very carefully. Schwartz and Dreyfus believe that very few patients recover. The treatment, unfortunately, is chiefly symptomatic. Absolute rest in that position which gives most easy respiration, should be provided. Hypnotics should be given to calm agitation. If the pneumothorax is increasing, puncture or a pleurotomy is indicated. A very grave emphysema may demand operation, which should be a pleurotomy. Hemothorax likewise may require intervention, the indications for which are the same as for wounds of the chest in general.

Perineal Extirpation of Cancers of the Anus and Rectum.—Delore and Chalier (*Revue de chir.*, 1907, xxvii, 784) say that the operation for artificial anus should be reserved for inoperable cases, and that these are being diminished in number as time goes on. Without passing judgment on the sacral method of Kraske, or the abdominoperineal operation, Delore and Chalier believe that the perineal route, or, better, the coccyperineal, suffices in the great majority of cases, if they are attacked early; and that it is available for the anorectal cancers or the cancers of the rectum proper, even the more extensive cases, such as the diffuse cases, the supra-ampullary, certain varieties of the rectosigmoid cancers. Delore and Chalier have operated on 19 cases, 2 of which were epitheliomas limited to the anus, 6 diffuse anorectal epitheliomas, and 11 cancers localized to the rectum (7 of the ampulla and 4 supra-ampullary); 2 were simply excised without amputation of the anus or rectum. The coccyperineal operation permits the wide and sufficient removal of the growth in the majority of cases, without the mutilation of the Kraske or the dangers of the abdominoperineal operation. A temporary artificial anus was performed in 1 case and gave perfect results. They did not employ it more frequently because of the difficulty in obtaining the patient's consent. They are not in favor of a permanent iliac anus, to obviate the difficulties of an incontinent perineal anus. They believe that the latter is better than a partially continent iliac anus. If the anal cancer is large and extends into the rectum, it will often be serviceable to curette and cauterize the external protuberant portion as a preliminary operation. This will permit more easy and thorough asepsis of the region and closure of the anus at the time of the operation.

The lithotomy position is employed. The incision surrounds the anus and extends forward in the median line a short distance, and backward to the coccyx, which is excised. The rectum with the invaded tissue and the sacral lymph glands are liberated *en masse*. This can be done up to the promontory of the sacrum posteriorly. The rectum is then liberated anteriorly, high up. The vagina can, with care, often be saved. By drawing down the mass the peritoneum is exposed anteriorly and opened with scissors, when gauze is introduced to protect the peritoneal cavity. The sheath of the rectum and the surrounding tissue are then divided, and by a sort of unsheathing of the rectum the mass can be brought down farther. The anterior cul-de-sac of peritoneum is then closed by suturing the cut edge of peritoneum in front to that of the pelvic colon drawn down. The intestine is then cut between forceps, the lower end with the mass is dissected out, and the upper cut end brought down and sutured to the skin about in the position of the normal anus. Iodoform gauze is used for drainage in front of and behind the rectum, and a rubber tube and gauze are introduced into the rectum. The operative mortality in the 19 cases was 10.5 per cent. So far as the late results could be ascertained they were very good as to duration of life. One woman, sixty-eight years of age at the time of operation, was free of recurrence eight years later.

Sarcoma of the Prostate.—PROUST and VIAN (*Annal. des mal. des organ. genito-urinaires*, 1907, xxv, 721) say that Langstaff and Bree, in 1802 and 1806, published the first 2 cases of sarcoma of the prostate. Burckhardt, in 1902, after a careful revision of preceding statistics, furnished a definite list of 24 cases of primary sarcoma of the prostate. Proust and Vian, by a search of the literature and the report of a new case bring the number up to 34. Of these 24, or 73 per cent., were under thirty years of age, and 15 of the 24 under ten years of age. While very rare between thirty and fifty years, it was more frequent beyond fifty years (7 cases out of 34). The symptoms vary markedly, according to the age of the patient; and Proust and Vian make two classes of cases, those under and those over thirty years age. In the old, retention rarely comes on suddenly, as it does in the young. In the old, the symptoms, aside from increased volume, are rather negative. The symptoms of dysuria are less marked than in hypertrophied prostate, catheterism is rarely difficult and is never impossible. The enlargement is much less marked than in the young and is rarely felt in the hypogastrium. It can always be felt in the rectum. In the young, the growth is so rapid that it is soon beyond the resources of the surgeons, while in the old, operation has obtained appreciable results, as far as the duration of life is concerned. The only operation advisable at the present time in the young is the suprapubic. The perineal exposes the patient to infection and sloughing of the tumor. Operation, even in adults, is very grave and difficult. Proust and Vian believe that for tumors of moderate size the perineal path should be selected, while for very large tumors or those projecting into the bladder the suprapubic should be preferred.

Experimental Substitution for the Mesentery.—LANZ (*Zentralbl. f. Chir.*, 1907, xxxiv, 617) says that in consequence of the fact that particles of the thyroid gland experimentally injected into the peritoneal cavity were found later wrapped up in omentum, he attempted to show whether and to what extent the attachmnt of the mesentery could be substituted by omentum, with preservation of the normal intestine. August 3, 1906, in a dog, he divided the mesentery between ligatures for about 3 cm. close to the small intestine. He then inserted a piece of the end of the omentum and sutured it to the whole circumference of the detached bowel. October 9 he reopened the abdomen and increased the separation of the mesentery from the bowel about 9 cm. farther and attached another portion of the omentum to the freshly separated bowel, not around the bowel, but simply by its free edge. November 9 the mesentery was separated 14 cm. farther and omentum substituted. November 30, 25 cm. more of the bowel was detached and attached to omentum. This made 50 cm. of bowel cut off from its normal blood and nerve supply, and now depending upon the substituted omentum. At the time of each operation the freshly treated bowel appeared as a contracted band of a bluish-white, mottled color, while that previously operated on had a normal color and its contractility was completely normal. On March 12, 1907, the dog died, when the 50 cm. of bowel had lived two and a half months, with its omental attachment. It had contracted to a length of 40 cm., while its calibre was narrower than its oral and caudal ends. The serous surface was smooth and shiny throughout.

Macroscopically there was no further difference between the affected and the normal bowel. The following practical application of this experiment was carried out by Lanz: A woman, aged sixty years, was admitted to the hospital, February 22, 1907. She had given birth to twenty-one children, was very senile and cachectic, and had a chronic intestinal obstruction supposed to be due to a cancer of the splenic flexure of the colon. Operation, February 27, showed a distended sigmoid flexure and volvulus and a cancer of the pylorus, which was resected. The transverse mesocolon was so adherent to the greater curvature of the stomach that about 6 to 8 cm. had to be cut through and for several centimeters the colon was completely detached from its blood supply. Resection would have been so dangerous that the omentum was brought up and its free edge employed as a substitute for the loss of mesocolon. April 19, 1907, the patient was in a favorable condition and her weight had increased from 39 to 46 kilograms.

A Simple Method of Operation for Stone in the Ureter.—Bartlett (*Zentralbl. f. Chir.*, 1907, xxxiv, 621) has proved by operation that stones of moderate size in the pelvic portion of the ureter can be easily located and removed by a new and very simple method. An incision is made through the abdominal wall at the outer border of the rectus muscle. The peritoneum, which is not opened, is displaced toward the median line, until the ureter closely bound to the peritoneum is found. The ureter and peritoneum are so loosely attached to the transversalis fascia that they are easily brought into view of the wound With the ureter grasped between the thumb and finger of the left hand it can be followed throughout its pelvic portion and the stone palpated. When the stone is found it is held between two fingers and pushed strongly against the wall of the ureter. A small opening is made by a sharp knife and the stone extruded and removed from the wound. The opening in the ureter is not sutured. A small rubber drainage tube is introduced to the region of the ureter and brought out of the lower angle of the wound, the rest of which is closed. In 3 cases this operation was completed, each time in ten minutes. The size of the stones varied from that of a bean to that of a hazelnut. In none of the 4 cases was a urinous odor detected in the dressing. The drain was removed in five to six days, and the 4 patients left the hospital, cured, within two weeks after the operation.

The Contributions of Surgery to a Better Understanding of Gastric and Duodenal Ulcer.—Mayo (*Annals of Surgery*, 1907, xlv, 810), from the standpoint of the operating-room results, divides ulcers of stomach into two classes: (1) The indurated or calloused ulcer which can be seen and felt from the outside of the stomach during operation, and (2) the non-indurated mucous ulcer which cannot be identified from the outside. All the positive advances in surgical knowledge concern the first class. Nearly all the failures of surgery are to be found in the second class. The relative frequency of ulcer has been placed at 10 gastric to 1 duodenal. In St. Mary's Hospital, from July 24, 1905, to March 23, 1907, 200 ulcer cases have been operated on. Of these 87 involved the stomach, 98 the duodenum, while 15 were indifferent ulcers of each viscus. The large majority of gastric ulcers involve the lesser curvature above the pylorus, and extend downward anteriorly and posteriorly in a saddle shape. The typical duodenal ulcer occurs in the upper one and a half inches of the duodenum, and in 96 per cent. of the cases extends upward to or within three-quarters of an inch of the pyloric sphincter. This has led to inclusion of these ulcers with the gastric. A thick-walled external vein best marks the position of the pylorus. The duodenal ulcer occurs well above the common bile duct, with its alkaline secretions. The clinical histories and pathological examinations of removed specimens made it certain that cancer had its origin in indurated ulcers. The topography of ulcer and cancer are the same. Cancer of the duodenum is a rare malady and its etiological relationship is apparently not important. From numerous experiences Mayo looks upon pyloric spasm as an indication of an irritation in some part of the intestinal tract which causes an irregular attempt to close the pylorus and thus prevent the food from entering the disturbed area. At the present time Mayo does not believe that a diagnosis of mucous or other undemonstrated ulcer indicates a surgical operation, unless complications, such as perforation, hemorrhage, or obstruction exist.

Partial Gastrectomy.—Frazier (*Annals of Surgery*, 1907, xlv, 950) calls attention to the large number of so-called cases of gastritis in which no definite lesion, such as ulcer, is diagnosticated, compared to the few which reach the hands of the surgeons. He reports a case in which, despite some negative findings, the diagnosis was made of carcinoma of the pylorus probably too far advanced for more than a palliative operation. At the time of operation it was decided to do a partial gastrectomy for the indurated area which was still considered to be a cancer. The patient recovered rapidly and three months after the operation had gained thirty-four pounds. The laboratory diagnosis was gastric ulcer. Frazier says that the lesson to be learned from the statistics he has studied is in the danger of placing too much reliance upon what may be called the refinements of laboratory diagnoses. He doubts that the mortality of excision of the pylorus for benign conditions will be greater than that of gastrojejunostomy. The expectation of life should be greater after the former operation, since the favorite seat, and a common predisposing cause, of carcinoma have been removed.

THERAPEUTICS.

UNDER THE CHARGE OF

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Amyl Nitrite in Hemoptysis.—Crace-Calvert (*Lancet*, clxxii, 4362) considers amyl nitrite to be most valuable in practically all instances of hemoptysis. At first it appears to be madness to give a patient, with a bleeding vessel, a drug which is a powerful vasodilator, but—even though its action be only transient—it produces such an immediate fall in the general blood pressure, that the pressure at the bleeding point is lowered and there is time for clotting to take place, and the hemoptysis usually ceases almost instantly. Amyl nitrite causes no reactionary pulmonary hyperemia, while adrenalin apparently does, and the former has the further advantage that it does not interfere with coughing and so places no obstacle in the way of the patient getting rid of the effused blood; hence, it lessens the risk of later septic trouble. He uses the drug in 3 minim capsules, one of which the patient breaks, inhaling the fumes as they arise. Warning should be given that the consequent feeling of fulness in the head is of no significance.

Antipyrine as a Sedative in Childhood.—H. N. Fletcher (*The Practitioner*, 1907, lxxviii, 540) considers antipyrine a powerful and valuable sedative in childhood. Its precise mode of action and the part of the nervous system which it influences are not known. It has not the dulling effect upon the cerebral cells which morphine, for instance, possesses. It does not appear to affect cerebral or motor activity to any extent, but rather to have some selective action in controlling sensory stimuli. He has observed no instance, in a series of several hundred patients, in which a rash or any other ill-effect followed the use of the drug. It is free from the hemolytic action which all the other members of this group unfortunately possess. The cyanosis, for instance, which may follow the excessive use of acetphenetidine or acetanilide, due to hemolysis and the formation of methemoglobin, does not result from the administration of antipyrine. It also has no injurious effect upon the metabolism of healthy tissues. If given in small doses (1 to 2 grains) its employment is almost entirely free from danger, and it needs but to be used to convince one of its efficacy and to cause one to consider it an invaluable ally in the treatment of the nervous disturbances of childhood.

Kephaldol: A New Antipyretic and Analgesic.—Einhorn (*Zentralbl. f. die Ges. Therapie*, 1907, xxv, 57) writes most enthusiastically concerning this new agent. Pharmaceutically it is a combination of citric and salicylic acids, phenetidine, quinine, and sodium carbonate. Its ordinary dosage is from 15 to 75 grains daily. It seems to have few disadvantages since it does not affect the heart untowardly, having the contrary effect of rendering the pulse fuller and stronger, it has no unpleasant action on the kidneys, it does not cause tinnitus, mental symptoms, or disorders of sight, and eruptions are not likely to follow its administration, nor does it disorder the digestive tract. As an analgesic he finds it valuable in the pains of syringomyelia, acute rheumatism, sciatica, neuralgias of various kinds, and even in those of locomotor ataxia. The pains of meningitis and cerebral tumor are unaffected by its administration. As an antipyretic it acts excellently in facial erysipelas and acute rheumatic arthritis. Furthermore, it possesses a marked anhydrotic effect, and when given in doses of 15 grains in the evening, is an active preventive of the night sweats of pulmonary tuberculosis.

Tuberculin in Ocular Tuberculosis.—A. Darier (*Revue de therap.*, lxx, 37) reports a number of instances of tuberculous ophthalmic conditions of various sorts—iritis, iridocyclitis, kerato-iritis, tuberculosis of conjunctiva, cornea, etc.—in which excellent results have been brought about by means of the injection of tuberculin. He admits that recurrences and relapses are not infrequent, but points out that ocular tuberculosis is a very grave condition and states that a recurrence is a signal for a renewal of treatment. The preparation used is the so-called tuberculin R. and the rule is to employ very small doses ($\frac{1}{500}$ mg. of the solid substance, 1 c.c. of the tuberculin R. containing 10 mg. of this) at first and if this causes a reaction this quantity is diminished. The injections should be undertaken with thorough asepsis and they only slightly painful. The site of injection may be between the shoulders or the region of the buttocks. The procedure should not be carried out oftener than every two days as a rule, but, when great care is used and the doses are small, daily injections may be employed, the quantity being very slowly increased. If the patient exhibits any rise of temperature the treatment should not be begun, and if the injections cause fever they should be stopped until this symptom has disappeared. If large doses (5 mg.) are employed they should be given not oftener than once a week; such a quantity is, however, seldom necessary. In the event of the employment of subconjunctival injections these should be given with the greatest care only and never before a long preliminary course of the ordinary hypodermatic injections.

Radiotherapy in Tinea.—Bureau (*Gaz. méd. de Nantes*, xxv, 157) considers that the *x*-rays are our most efficient means of treatment in ringworm of the scalp; they accomplish cure in three or four months while other methods often take two or three years. The technique is as follows: Each lesion is exposed to the rays, in a position six inches distant from the centre of the tube. The time of the exposure is that necessary for a pastille of barium platinocyanide placed at a distance of about three inches to assume the color "B" of the radiometer of Sabourand Noiré. The exposed region becomes entirely epilated in from fifteen to eighteen days. The hair begins to grow again, about eight to ten weeks later, and is complete in from three to four months.

The Iodides in Cardiac Conditions.—Barié (*La quinz. thérap.*, 1907, vii, 513), during the period of loss of compensation in cardiac disease, advises the use of digitalis, but after compensation has been re-established prescribes the iodides. These are advantageous from three standpoints: their vaso-dilator action, their effect in lowering blood pressure, and their so-called antisclerotic power. The elimination of the iodides is rapid and takes place through various channels and, although the treatment may be continued for months, iodism seldom results. Certain patients, however, possess an intolerance which is impossible to conquer. The iodides are indicated in arteriosclerosis, angina pectoris, and coronary arteritis, as well as in valvular affections. They are also beneficial in affections of the heart muscle, such as myocarditis and the succeeding sclerotic conditions. Especially are they useful in chronic aortitis and aortic aneurysm. The salts of potassium and sodium are most frequently prescribed, the former if a syphilitic element is present, the latter being preferable under other circumstances since it is more easily assimilated and is less likely to disturb the stomach. The administration of the iodides may be continued for considerable periods with intermissions during which tonics, such as iron, arsenic, or quinine, should be prescribed.

Sterility and the X-rays.—BERGONIÉ and TRIBONDEAU (*La revue medico-sociale*, 1907, i, 31) have proved that the *x*-rays exercise an intense destructive action upon the spermatic cord. A single exposure may be sufficient to sterilize the seminal canals in the animal, although the general condition remains unaffected and the sexual ardor continues unimpaired, even when the semen is deprived of spermatozoa. Other observers have confirmed these results and consider that sterility is very easily caused in man by *x*-ray exposure, owing to the situation of the testes and the slight protection afforded by the scrotum to these organs. In the female, the ovaries being deeply situated, sterility is less easily brought about. Those who work with the Röntgen rays should take the very necessary precaution of wearing shields of lead or of rubber covered with bismuth.

Opsonins and Their Relations to the Treatment of Bacterial Infection.—OPIE (*Yale Med. Jour.*, 1907, xiii, 301) asserts that the early work of Wright and Douglas has shown that normal blood serum exerts an influence upon phagocytosis of certain bacteria. The power of the serum to promote phagocytosis may be increased by immunization. Injection of a bacterial vaccine results in a phase of decreased opsonic power followed by a phase of heightened activity. It is not yet possible to decide with how great success Wright has applied these facts to the treatment of infections. The determination of the so-called opsonic index is subject to grave error, but at present offers the best means of estimating the power of the blood serum to promote phagocytosis. In spite of errors determination of the opsonic index indicates the occurrence of a negative phase of opsonic power following inoculation and offers the only possibility of avoiding the harmful effect of decreasing by such inoculation, rather than increasing, resistance to bacterial invasion. The opsonic index with the affections which have been examined, and notably with tuberculosis, is subject to such wide variation that it promises at present little diagnostic aid. The almost uniform success claimed by Wright and his co-workers in treating suppurative infections and localized tuberculosis is sufficient warrant for the widespread interest which the subject is attracting.

Contra-indications to the Use of Opium in Acute Disease.—BOUSFIELD (*Pract.*, 1907, lxxviii, 663) states that there are certain contra-indications to the administration of opium in acute diseases; some precluding its use altogether while others suggest that only small doses may be given with safety. If the lungs are embarrassed with a considerable amount of fluid arising from any cause, as met in the œdematous bases often observed in acute disease in old people or in those suffering from heart weakness, opium should be avoided, any ease resulting from its use being too dearly purchased. The susceptibility of young children to the drug as a rule precludes its use in their acute diseases, except in such conditions as cholera infantum, in which its specific action on the intestines may be valuable. In older children opium should be given in moderate doses only, and its effect should be carefully watched; the same is true in respect to aged patients. With regard to high arterial tension and other signs of chronic nephritis, the fact that opium is excreted almost wholly by the bowel and scarcely at all by the kidneys, and that it may be given in uremia with safety, might show that this condition might not contra-indicate the use of opium; this may be the case when the disease is strictly localized, as in some limited acute inflammation or spasmodic contraction of a muscular tube, but when the acute disease is of general or infective character there is certainly, as in instances of chronic renal disease, a special danger that respiratory complications, such as diffuse bronchitis or pulmonary œdema may supervene; consequently opium should be used with great caution, if at all. Digestive disturbance, headache, and marked constipation are in lesser degree, contra-indications. In any acute abdominal disease, if the question of operation is to be raised, care must be taken to give opium in such moderate doses that, while immediate pain is relieved, the clinical picture is not obscured.

Basic Quinine Acetyl-salicylate.—Santi (*Les nouveaux remèdes*, 1907, xxiii, No. 5) has been able to prepare this salt, which he expects will advantageously replace quinine salicylate, as follows: He mixes equimolecular amounts of acetyl-salicylic acid and quinine hydrate and dissolves them in ether, following which an oily precipitate forms, which soon becomes a crystalline mass; this is basic quinine acetylsalicylate, a bitter, white salt, slightly soluble in water, more soluble in alcohol and chloroform and almost insoluble in ether. Experimentation seems to show that this salt is of value in pleurisy and peritonitis and that it is a more effective antipyretic than the ordinary quinine salts. The ordinary dose is 6 grains.

Quinine Phytinate.—According to Posternack (*Jour. de méd. de Paris*, 1907, XIX, 88) this substance is an anhydro-oxymethylene-diphosphoric acid of quinine containing 57 per cent. of quinine and 43 per cent. of phytinic acid; consequently, it allows the administration of these two agents each in sufficient dose. Quinine phytinate is indicated in all conditions in which the ordinary salts may be employed. Phytinic acid is a valuable and certain general tonic to nutrition and is an aid to the organism in combating microbic infection, consequently it is a useful adjunct to quinine in its action upon the malarial organism and in puerperal and enteric fevers. Quinine phytinate is especially indicated in malarial cachexia and it has been employed with good results in the neuralgias and vertigo of Ménière's disease; and in certain forms of diabetes it is considered to affect favorably the destruction of sugar in the organism.

Thyroid Extract in Hay Fever.—Pottier (*Jour. de méd. de Paris*, 1907, xix, 130) has employed thyroid extract in three instances of hay fever. In one of these a complete cure was established, in another the result was unsatisfactory on account of co-existent affections, such as hemorrhage into the vitreous body due to arteriosclerosis; in a third the patient suffered from more of the symptoms of his disease through a long and particularly hot summer even though he was exposed to the usual influences which had been accustomed to give him trouble. The dosage prescribed was 1½ grains of the extract given twice daily, with periods of intermission. While Pottier states that the treatment has been employed in too few patients to draw definite conclusions, he considers that this form of treatment merits further trial.

The Treatment of Angina Pectoris.—WEISSBART (*Zentralbl. f. die gesammte Thérapie,* 1907, xxv, 173) reports upon a preparation known as dyspnon which is dispensed in tablets containing each 3¾ grains of theobromine sodiosalicylate, 1½ grains of theobromine sodio-acetate and 1½ grains of extract of quebracho, as being of value in angina pectoris and allied conditions. He has used dyspnon in five patients. The first was an instance of syphilitic aortic insufficiency with coronary angina in which the anginoid attacks remained absent as long as the medication was continued. Good results also followed its use in an instance of stenocardia due to aortic and mitral insufficiency and in a patient suffering from arterioscerosis with apoplectic seizures. Weissbart recommends the employment of this preparation in the cardiac asthma of endocarditis and arteriosclerosis. The usual dosage is two tablets three times daily

The Various Carbohydrates in Diabetes.—LABBE (*Revue de thérap.,* 1907, lxxiv, 229) considers that the proper selection of carbohydrate foods for the diabetic is quite as important as any other point in the diet regulation of this disease. All carbohydrate foods do not by any means act the same upon these patients. In accordance with the tolerance of the diabetic for carbohydrates Labbé arranges this class of food in the following order: Potatoes, oatmeal, macaroni, chestnuts, rice, dry beans, lentils, dry peas, milk, bread, sugar. Diabetics are able, without exhibiting glycosuria, to eat a larger quantity of potatoes than of bread, and the former article of food is the carbohydrate of choice for patients suffering from this disease. Cereals, rice, and dry legumes are almost equally good, but bread should be forbidden. The difference in the toleration of diabetics for the various foods of this class is due to the difference in their chemical constitution and to the intra-organic evolution of the different starches and sugars.

Thyroid Extract in Cretinism.—FISCHER (*Monthly Cyclopedia of Pract. Med.,* 1907, v, 210) cautions against the toxic effect of an overdose of this substance, which has such a remarkable effect in cretinism. The physiological effect of the extract should be studied exactly as we should study the effect of drugs in any instance in which circulatory disturbance exists. The stimulant action of the thyroid extract upon the vascular system often causes flushing of the face, marked restlessness, and rapid pulse rate. We should always be on the watch for peculiar susceptibility and idiosyncrasy regarding thyroid treatment. Five grains of the desiccated gland may be given daily, which is the equivalent of one-sixteenth of an average sheep's thyroid gland. Some patients will respond to half this dose, while others may require a larger quantity. The first evidence of overdosage is a gastro-enteric disturbance, with vomiting, diarrhœa, and fœtid breath. There may be abdominal pain and tenderness and very rapid loss of weight. Blood examination will show marked destruction of the red corpuscles.

Electric and X-ray Treatment of Neuralgia.—De Courmelles (*Bul. gén. de thérap.*, 1907, x, 367) considers that faradism is inferior to galvanism in the treatment of neuralgia. For facial neuralgia the positive pole is applied to the painful area for from five minutes to an hour, while in sciatica the negative pole is placed over the seat of the pain. The author finds also that the ultra-violet ray acts as a sedative, while the blue light is less active in this regard. The *x*-rays and radium are also very useful, but must be employed with skill and care. Their action is rapid, the pain being lessened in a few minutes and, if the treatment is continued for several (twelve) sittings, may be wholly relieved. The treatment should not be given oftener than once daily if the patient exhibits the least febrile reaction.

Renal Decapsulation in Nephritis.—Ertzbischoff (*Thèse de Paris*, 1906; *Revue de thérap.*, 1907, lxxiv, 176) has studied the effect of this operation in 15 patients whom he has been able to follow. In a few of these death succeeded the operation; the fatal outcome was the result of uremic poisoning. In 6 patients the result was *nil*, the albuminuria persisting as before. In 2 patients only was there real improvement; here with the aid of a diet lacking in chlorides the albuminuria diminished or disappeared. On reviewal of these and other previously published instances of renal decapsulation we can but realize that the procedure has hardly given the good results hoped. However, it would seem that it is of benefit in the so-called infectious nephritis and that the contrary is true of nephritides of intestinal origin; here the operation may be undertaken, not with the hope of cure, but with the expectation of lessening the hematuria and the pain. An anatomic study of the kidneys of the deceased patients shows that a new and thicker capsule is formed and that a secondary sclerosis takes place which may remain superficial or extend into the substance of the organ.

Santonin.—Sejournet (*La quinzaine thérap.*, 1907, viii, 31) has studied the interesting question of the treatment of diabetes by means of santonin, and states that under its influence the glycosuria becomes rapidly diminished, the polyuria becomes lessened, the patient's strength is increased, and the thirst and the dryness of the mouth are ameliorated. Santonin also possesses a stimulant effect upon the nervous system as well as an antispasmodic action, and, in view of the important role played by this system in the genesis of diabetes, ought to diminish the glycogenic function of the liver. Santonin should be prescribed, just as are valerian, camphor and its bromide, and the various cyanides, in conditions in which we wish to lessen or prevent excessive muscular contraction and when we desire to restore to the nervous system its normal, regulator action. Bircage considers that this drug possesses an analgesic effect in the lightning pains of tabes and that its feeble toxicity permits its continued administration.

The Internal Treatment of Acne.—Kapp (*Thérap. Monatsh.*, 1907, xxi, 138) has found what he considers a distinct relationship between indican and other products of the putrefaction of proteid in the intestinal tract, in the urine, and acne. This connection naturally suggests combating the intestinal condition as a means for the relief of the folliculitis. Kapp prescribes as an intestinal antifermentive a combination of 15 grains of precipitated sulphur and 4 grains of menthol; this is taken two or three times daily. A series of 33 patients affected with the various types of acne, simplex pustulosa, and indurata, have received this treatment for from three to four months with excellent results. The only unpleasant effect of the treatment was one instance of temporary diarrhœa and coliky adbominal pain; under its employment the products of intestinal putrefaction in the urine became much diminished and the acne was markedly benefited. With the internal medication the usual local treatment was prescribed.

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.,

OF PHILADELPHIA.

Impaired Resonance Behind and Beneath the Inner Third of the Clavicle in Normal Children.—The areas of impaired resonance S. McC. Hamill (*Archives of Pediatrics*, 1907, xxiv, 92–94) found in the chests of the majority of infants and children in perfect health. He elicited it with greater difficulty in infants than in older children; he found it less common in later childhood than in the early and middle periods. In the majority of the children it did not extend beyond the inner third of the clavicle; it is elicited by percussing the clavicle and the first interspace immediately beneath. Sometimes it extends as far as the midclavicular line; it then extends downward, blending into the upper border of cardiac dulness. It is more easily noted with the child lying down than in the upright posture. He explains it by asuming that the posterior position of the lung in early life brings the great vessels into more intimate contact with the anterior chest wall.

A Case of Knotting of the Hair.—J. M. H. Macleod (*Brit. Jour. Dermat.*, 1907, xix, 40) reports the case of a girl of thirteen, afflicted with trichonodosis. Her hair would not grow properly, breaking off whenever it reached her shoulders. At the age of six, during a pneumonia, to facilitate the ice applications her hair was cut; as it grew badly during covalescence her scalp was shaved to stimulate the growth of hair; but in spite of all treatment it never recovered. The ends of the hairs were either split into several fibers, pointed and atrophic, or bent up like a hook. There was no pityriasis, no hyperkeratosis at the mouth of the hair follicles, or any other pathological change. The hairs were dry, lustreless, but pigmented; the majority formed loops at the end. Some were twisted on their long axis and formed single or double loops or knots toward their periphery; some of the knots were drawn tight, others loose. No organisms or fungi could be discovered on careful examination. The mode of development of this curious malformation could not be discovered.

The Etiology of Convulsions in Infancy and Childhood.—James McIlraith (*Medical Chronicle*, 1906, November and December; 1907, January) has studied 250 cases in which convulsions occurred. The two chief predisposing causes are an hereditary history of neuropathic taint (143) and rickets (94); both causes co-existed in 25 of the cases. The exciting causes were as follows: abnormalities of labor 10, cerebral paralysis 8, infantile paralysis 2, congenital syphilis 6, hydrocephalus 2, meningitis 1, trauma 9, insolation 1, dentition 3, gastro-intestinal disorders 108, overloading stomach or improper food 7, reflex irritations from various sources 20, onset of measles 7, onset of pneumonia 13, onset of rheumatic fever 1, whooping-cough 7, during measles or pneumonia 4, following an acute fever 9, jaundice 3, mental deficiency 5, idiopathic 24. His conclusions from the study of the cases are: Convulsions are most frequent during the first year of life, and not very frequent after the third year. The predisposing causes are more important than the exciting causes. The predisposing causes are an inherited neurotic taint and rickets. Healthy children born from healthy parents rarely suffer from convulsions. In children suffering from convulsions there is frequently a history of ill-health or disease in the parents, especially in the mother during pregnancy, and this acts by lowering the vitality of the child and rendering it more liable to disease. The family history of children suffering from convulsions is bad. Frequently the brothers and sisters suffer, and the mortality in these families is high. Only a small proportion of cases can be ascribed to organic disease of the brain or to injuries at birth. The most common cause of convulsions in the first two months of life is reflex irritation from the alimentary tract. The great majority of cases are due to reflex causes. Dentition is rarely a cause of convulsions, and only when some predisposing cause exists. Gastro-intestinal disorders are the chief exciting causes of convulsions. Convulsions are by no means as frequent as generally supposed at the onset of acute fevers, such as measles and pneumonia. They are more common at the onset of pneumonia than at the onset of measles. When they do occur there is usually some predisposing cause present. In several of the cases of convulsions associated with whooping-cough a predisposing cause was present. Convulsions are by no means frequent in whooping-cough, when no predisposing cause exists and no complications occur. Convulsions in early life may be the first sign of epilepsy, or may give rise to that condition in later life, and this is more likely to occur when there is no obvious cause for the attack. The fundamental cause for convulsions is some predisposing cause.

Bronchitis in Children.—The treatment of this condition is outlined thus by John U. Fauster (*Medical Council*, 1907, April, 132–135): The hygienic part consists in keeping the temperature of the room uniform, between 68° and 72°, during the twenty-four hours; sunlight is considered desirable. The child should be kept in a bed, so it can be moved about from time to time; if the room is draughty a screen should be kept around the bed. Unnecessary noise and visitors should be excluded. An extra-long flannel night-gown should be worn, so that the patient may wriggle about. The food should be given frequently and in small amounts; it should be of the blandest character. Water should be given frequently by the mouth, but Fauster does not approve of sponging, as it contracts the superficial capillaries and still more engorges the already impeded circulation. He advises a mustard plaster to the chest, this to be kept applied until full dilatation of superficial capillaries is produced; the chest should then be covered with a cotton jacket; this procedure must be repeated every twenty-four hours. The author disapproves of the various types of poultices, clay applications, and inhalations. Medicines should be of pleasant taste, as otherwise the worry and excitement attendant upon their forcible administration produce positive injury. The bowels should be well opened; the best drug for the purpose is in calomel, $\frac{1}{10}$ grain every hour until the bowels are thoroughly evacuated. Of other drugs he considers ipecac indicated when there is troublesome cough with whistling rales; belladonna, when the secretion is very abundant and a respiratory stimulant be needed; aconite, if there is fever and the heart overactive; bryonia, when there is a dry, painful cough and danger of extension to the lung parenchyma exists. The drugs should be given in small doses frequently repeated; they may be given singly or in combination.

Maniacal Chorea.—J. Magee Finny (*Brit. Med. Jour.*, 1907, i, 976) reports a case of this condition in a girl of seventeen. She had been complaining of rheumatic pains in the legs for several weeks; they disappeared and chorea supervened. A mitral systolic murmur was audible from the beginning, of varying loudness and lasting up to the ninth day—the day of her death; the pulse was quiet and regular each day (about 80) until the last three days, when it rose to 140; the temperature was normal until the day before death, when it reached 104°. The choreic movements were slight for the first two days, and then they became so violent that two nurses were required to watch her, to prevent her from falling out of bed and injuring herself. The psychic phenomena were out of all proportion to the motor, and at first overshadowed the motor; they differed from the acute delirium of fever and acute mania by the absence of incoherent speech and wild garrulity. There were occasional periods of mental calm, during which she ceased shouting and her movements became quieter; but these lasted but a short time. Medicines were of no service beyond giving an occasional period of sleep; chloral and chloroform inhalations were the most valuable for this purpose. The autopsy revealed no abnormality beyond hyperemia of the brain and a few thromboses of small vessels of the cortex. There was no mitral valve disease and the murmur must have been due to ventricular dilatation. No microorganism was found either in the cultures made from the spinal fluid or in any of the sections of the meninges.

Multiple Neuritis (Non-diphtheritic) in Children.—H. M. Thomas and H. S. Greenbaum (*Jour. Amer. Med. Assoc.*, 1907, xlviii, 1397) have been able to collect and tabulate from the literature 138 cases of non-diphtheritic multiple neuritis in children. Measles was claimed to have been the cause in 8, whooping-cough in 7, scarlet fever in 6, and 27 were due to other infectious conditions; of 48 toxic cases 19 followed lead and 19 arsenic chorea; in 33 the cause could not be determined. The condition is usually considered as very rare, but the authors believe it to be less uncommon than usually thought. They have seen 10 cases; 7 were in girls and 3 in boys; the ages varied from 3 to 12. In 3 the cause was found to have been typhoid fever, in 2 lead, in 1 arsenic and chorea (fatal), and in 4 the cause could not be determined. The cases are reported in detail. It is seen that the symptomatology does not differ materially from that of the disease in the adult. Sensory disturbances are said to be difficult to determine, and when present do not persist very long; reactions of degeneration with atrophy are generally noted; the deep reflexes are lost during the height of the attack, but return during recovery. The paralysis is usually widespread and symmetrical, affecting both upper and lower limbs; the legs are generally more severely involved than the arms. The differential diagnosis from poliomyelitis is not always easy: multiple neuritis comes on more insidiously, its cause is often apparent, it rarely is associated with fever at the outset, objective disturbances are more common in neuritis, the paralysis is more widespread and symmetrical, and recovery is more common. The prognosis in multiple neuritis of children is usually good, but few deaths having been recorded.

OBSTETRICS.

UNDER THE CHARGE OF

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Two Cases of Repeated Ectopic Gestation.—Kynoch (*Jour. Obstet. and Gyn. Brit. Empire*, December, 1906) has collected 585 cases of ectopic gestation, among which there were 34 cases of repeated extra-uterine pregnancy. No authentic case of ectopic pregnancy occurring three times in the same patient has yet been reported. The most frequent form of reptition is in the opposite tube.

Kynoch's first case was that of a woman married fourteen years, and having six children without miscarriages. Menstruation had been regular except during pregnancy. The last normal menstruation was ten weeks before admission to the hospital, and for a month before coming under observation the patient had complained of intermittent pain in the left iliac region, with irregular hemorrhage. The pain came on suddenly and was severe, and made worse by defecation.

On examination the uterus was somewhat fixed and displaced by a swelling in the left side of the pelvis. Under anesthesia this was made out to be about the size of an orange, and in the left broad ligament. It was considered a hematoma as the result of a ruptured tubal pregnancy, and operation was deferred to await absorption. After ten weeks in the hospital the patient left in good health.

She was again admitted to the hospital about eighteen months afterward complaining of pain in the right iliac region. A tender swelling was found at the right side of the uterus, and upon operation the right tube was found adherent, and removed. The left uterine appendages were also adherent, and there was thickness in the upper part of the broad ligament.

On examining the specimen removed decidual cells were found, establishing a diagnosis of ectopic gestation. The case proved to be a six weeks' ectopic gestation in the left Fallopian tube, rupturing into the broad ligament and becoming absorbed in two months, followed eighteen months afterward by pregnancy in the right Fallopian tube.

The second case had at first an ectopic gestation upon the right side, treated by operation. The right tube was distended with blood clot, and was removed. The left tube and ovary were normal. The patient made a good recovery from operation and was subsequently delivered by forceps of a healthy male child.

Her second ectopic pregnancy developed about eighteen months afterward in the left tube, and upon operation a clot the size of an orange was found surrounding the fimbriated extremity. This was removed and the patient made a good recovery.

The Pathology of Cornual Gestation.—Lockyer (*Jour. Obstet. and Gyn. Brit. Empire,* December, 1906) describes a specimen obtained by Doran of cornual gestation ending in rupture. Microscopically the ruptured cornu was of nearly square shape, with a laceration extending the whole width of the upper border. The wall of the gestation sac varied greatly in thickness, the fœtus lying in a closed amniotic sac, covered in places by adherent blood clot. The placenta in its formation was very diffuse. The fœtal side of the placenta was smooth, represented by chorion from which the amniotic sac had been stripped during the expulsion of the fœtus. The pedicle of the gestation sac was solid, containing large vessels with thickened walls.

Nothing resembling a uterine mucosa or basal decidua was found in the specimen. The villi of the chorion were planted directly on the uterine muscle by masses of Langhans' cells. Where this came in contact with the muscle there was an inflammatory area mixed with cells of fœtal origin, forming an infiltrating zone. Just beneath this the muscle was in cloudy swelling. The appearances were those of ordinary inflammation.

The walls of the pregnant cornu were thicker than the walls of a pregnant Fallopian tube. The eroding action of the ectodermal tissue could be very plainly studied. The placenta was attached to the whole surface of the sac wall, and rupture occurred through the upper pole where the rudimentary horn is thinnest. In such tissue there is no retraction and relaxation, and hence the point of rupture. No vestige of normal glandular mucous membrane was found, and no decidua. The villi were dissecting the muscle layers, opening vessels and lymphatics. This resembled a malignant process with total absence of decidua. By repeated sections through a uterine gestation, it has been observed that the vesical of the embryo and its chorionic offshoots do not approach the muscular tissue of the uterus. In the gravid cornu the villi attach themselves directly to the muscular tissue by clusters of the ectodermal cells; while in the normal uterus the ovum is embedded in mucous membrane, in the rudimentary horn, the ovum seeks nutrition directly from the muscle. If the horn of the uterus be well developed the pregnancy will go to full term; if feebly developed, the muscular wall will rupture. The weakest point in the muscle wall against invasion by fœtal ectoderm is found by microscopic study to be the vessels. This is illustrated by microscopic drawings. The muscle fibers fail to form a compact decidua, the characteristic decidua being never evolved from muscle cells. After the rupture of the cornual sac the thinned-out muscular tissue is thrown into convolutions, the clefts of which are filled with fibrinous material from lymph and blood clot. The muscular convolutions form before rupture and are caused to rupture by the contraction of the muscular tissue. Over the weakest point in the gestation sac was found a covering of lymph with extravasated blood. This had probably exuded before the escape of the ovum. The latter occurred during the examination of the patient in the clinic by bimanual palpation. This has repeatedly happened in the experience of other observers.

Ectopic Gestation at Full Term, with Abdominal Section.—TATE (*Jour. Obstet. and Gyn. Brit. Empire*, December, 1906) reports the case of a patient admitted to St. Thomas' Hospital in her first pregnancy. Some time previously, when about three months pregnant, the patient had an attack of severe pain in the lower abdomen, continuing for several days, and in a lesser degree for about a month. This pain returned, and the patient was in bed about two months. During the early part of the day, while in pain, blood clots were passed from the vagina. Since leaving her bed the patient felt weak, with abdominal pain, and the abdomen increased in size. Fœtal movements were recognized for about six weeks, and then ceased.

False labor developed, which subsided, recurring a month later. As normal labor did not develop, the patient went to the hospital, where a diagnosis of ectopic gestation was made.

On examination, the fœtus could be plainly felt, made out in an abdominal tumor, extending about two inches above the umbilicus. The uterus could be recognized as not in the tumor, and the fœtal heart could not be heard. On abdominal section the dead fœtus was found free in the peritoneal cavity. The omentum and intestines were stained with meconium; the cord extended to a large elastic swelling in the lower abdomen, formed by the placenta, Fallopian tube, and broad ligament. This mass was removed entire by a series of ligatures, and vessels, in addition, were tied. There was considerable hemorrhage during enucleation of this mass. The patient was given intravenous saline transfusions and at once improved. She ultimately made a good recovery. On examining the fœtus, it was found at full term without offensive odor, and the umbilical cord was attached to the placenta at the edge.

Intraligamentary Pregnancy Going to Full Tetm.—Lyle (*Jour. Obstet. and Gyn. Brit. Empire,* December, 1906) reports the case of a patint who had suffered from free hemorrhage, continuing for several months. Amenorrhœa developed, and the patient found herself pregnant. A tumor developed at the right side of the abdomen, and one also upon the left. Although the patient usually had vomiting during pregnancy, it was absent in this instance. Upon examination, a tumor was found upon each side of the uterus at the brim of the pelvis. A uterine souffle was heard below and upon the left of the umbilicus; there were no heart sounds. Upon operation a tumor was found in each broad ligament which contained an ectopic gestation. The gestation sacs were both removed.

Lyle also reports a second case of intraligamentary pregnancy in which abdominal symptoms of pregnancy developed, with fœtal movements, which ceased after the patient had a serious cardiac attack. The patient's symptoms gradually subsided. The abdomen was enlarged and about the size of a seven months' pregnancy. There was no pain or tenderness, and no fœtal heart sounds were heard. Upon operation the tumor resembled a pregnant uterus. The wall of the sac was muscular, the sac containing a macerated fœtus. It was impossible to remove the sac, as the effort to do so produced free hemorrhage. The sac was thoroughly washed out with normal salt solution, the wound carefully sutured, and the abdomen closed. After the operation the patient had fever, and on the eighteenth day the sac was opened through the vagina, followed by the discharge of pus and gas. Under douches, the temperature fell to normal, and the patient made a good recovery.

Death from Hemorrhage and Septic absorption after Section for Full-term Ectopic Pregnancy.—Fairbairn (*Jour. Obstet. and Gyn. Brit. Empire,* December, 1906) operated upon a patient having a tender abdominal tumor, which was thought to be a dead child possibly retained in the uterus. Under anesthesia the uterus was explored and found to be empty and lined with decidual membrane. Upon opening the abdomen, a dark, cystic tumor was found which seemed to be behind the peritoneum. The intestines had been lifted up, and lay behind the sac. After packing around the sac, it was opened and contained a dark fluid with an inoffensive odor. The placenta was attached to the lower wall of the sac, and the attempt to detach a small portion of the edge caused serious bleeding. This became dangerous, and was controlled by packing. The sac wall was sutured to the abdominal incision.

The patient continued to bleed at intervals, and gradually symptoms of septic infection developed. On again exploring the cavity a piece of very offensive placenta was removed, followed by free hemorrhage. This seemed to cause the death of the patient. At autopsy peritonitis was absent and the sac was in front of the peritoneal cavity with a very extensive base, the pregnancy having originally developed from the right broad ligament. A large placenta in a partially broken down condition was found.

This case illustrates how difficult it is to trace the entire sac of an abdominal pregnancy, and how persistent is the circulation in these cases when active hemorrhage occurs six weeks after the death of the fœtus. Cultures taken from the sac were practically sterile.

Full-term Pregnancy in a Bicornate Uterus, with its Removal.—ROBERTS (*Jour. Obstet. and Gyn. Brit. Empire*, December, 1906) reports a remarkably interesting case in which a fœtus went to term in a rudimentary horn of a bicornate uterus. The fœtal sac did not rupture; the fœtus died and was retained in the abdomen for six months. During this pregnancy the patient was almost as well as usual, complaining of very little except the inconvenience caused by the tumor. Upon operation the tumor was covered by adherent omentum. It had a grayish-brown appearance, often seen in ovarian cysts which are degenerated. In trying to lift up the tumor the adhesions had to be separated. Its attachments or base were the left tube and ovary, where good-sized vessels had to be divided before the tumor could be removed. The right tube and ovary were normal. The pedicle of the tumor was the remains of the left tube and a portion of the broad ligament. The patient made a good recovery.

A similar case is reported by Mauriceau. In describing it Mauriceau distinguished plainly between tubal and cornual pregnancy, basing his distinction on the relations between the round ligament and the gestation sac. The condition was discovered at an autopsy in the seventeenth century, in a woman who died suddenly after abdominal pain, which was intense for three days.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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Heredity in Myopia.—WORTH (*Brit. Med. Jour.*, February, 1906) analyzes 687 cases of myopia, of which 33 were malignant, with reference to heredity. This factor was present in 56 per cent. of the non-malignant, and in only 21 per cent. of the malignant cases. In one family all the males were myopic, the females not, but it was through the latter that the myopia was transmitted. All the non-myopes had blue irides, the myopes brown. Of the 371 hereditary cases the parents were affected in 159; in the remainder it was the uncles, aunts, and grandparents who were myopic.

Iridectomy Combined With Sclerectomy in the Treatment of Chronic Glaucoma.—Lagrange (*Archiv. d'ophtalmol.*, August, 1906) has advised a new procedure for the establishment of a filtration cicatrix. An incision is begun with puncture and counter puncture in the sclera as near the angle of filtration as possible. Just as the section is about to be completed the edge of the knife is turned a little backward, the result of which movement is to produce a bevelled opening in the sclera like the vents of a flute; when the knife has cut through the sclera and has come to lie beneath the conjunctiva, it is turned directly backward so as to detach a large conjunctival flap. A small bit of that portion of the scleral flap toward the cornea is excised; to accomplish this the conjunctiva is raised by a narrow clawed forceps curved on the flat. The anterior lip of the wound thus becomes prominent and a narrow flap can be readily cut from the sclera by very sharp fine scissors sharply curved. A wide iridectomy as usual completes the operation. After healing is complete, a narrow white line beneath the conjunctiva marks the site where the sclera has been exsected. Lagrange claims that this sclerectomy provides an adequate exit of filtration. He has practiced this method upon twenty cases, without serious difficulty or the least immediate or consecutive accident. The results are stated to have been quite satisfactory. Fifteen cases are fully reported and commented upon, of which the oldest goes back three years and none less than six months.

He adds that while an eye affected with angiosclerosis will necessarily continue to undergo the process of sclerosis, and that if the nerve is undergoing atrophy under the influence of a diathesis, such atrophy will necessarily continue, he is entirely convinced that the filtration scar obtained by this procedure possesses all the advantages of a cystoid cicatrix without the disadvantages of prolapse of the iris. Hypertension is abolished, than which nothing more fortunate can occur in the case of an eye affected with glaucoma.

Arterial Tension in Cataract.—Fraenkel and Garipuy (*Archiv. d'ophtalmol.*, October, 1906, p. 615), based upon personal researches, have come to the following conclusions as regards the arterial tension in senile cataract: (1) Senile cataract is accompanied but rarely by arterial hypertension. Of 99 cases it was found in only 9. (2) Hypertension is regularly found in those affections which it usually accompanies without reference to the presence of cataract (nephritis, diabetes). (3) The age has no influence upon the arterial tension. (4) If it is true that arteriosclerosis is always accompanied by a certain degree of arterial hypertension, it may be affirmed that arteriosclerosis does not usually accompany cataract and that the former is not a factor in the causation of senile cataract. (5) The decrease in renal permeability which is found in those affected with cataract has no appreciable influence upon the general circulation. (6) It may be addmitted that diminution of renal permeability, too slight to give any appreciable clinical symptoms, suffices for the retention of crystalline cytotoxins within the organism. (7) Arterial hypertension in senile cataract contributes to the production of postoperative intra-ocular hemorrhages, either into the anterior segment of the eyeball or from the choroid (expulsive hemorrhages). (8) It is therefore possible to recognize before the operation that small number of patients who, by reason of arterial hypertension, are exposed to such hemorrhages, and so to permit of efforts being made to forestall such complications in diminishing the tension by proper therapeutic means (mode of life, purgatives, iodides, etc.).

So-called Rheumatic Diseases of the Eye.—Antonelli (*Archiv f. Augenheil.*, August, 1906, p. 404) maintains that the term "rheumatic" should be abandoned in ophthalmology. Embraced under this term various disease processes have been included which have nothing in common. Ocular diseases (uveitis and retinochoroiditis) following acute arthritis or polyarthritis are of infectious or infectiotoxic origin. Those occurring in pseudorheumatism belong to syphilis, tuberculosis, or gonococcic infection. Of those arising after catching cold, iritis rheumatica, scleritis, keratitis, episcleritis, and tenonitis in subjects affected with chronic rheumatism, it is not yet proved that all are due to the diathesis in question.

The ocular diseases ascribed to "arthritism" are in part due to autointoxication, as, for example, many forms of chronic blepharitis or blepharoconjunctivitis, and in part to gout, as for example many varieties of retinochoroiditis.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Wood Alcohol Poisoning from Varnish—An unusual case of poisoning by wood alcohol is reported by H. Gifford (*Ophthalmic Record*, June, 1906). After working about four hours in a room of 1800 cubic-feet capacity, tightly closed so as to keep out dust, applying to the woodwork first a stain and then a varnish, both made with wood alcohol, a man became absolutely blind, but had no other symptoms of poisoning. After a week, vision began to return, but after a few weeks it began to fail again, and in six months he was quite unable to see. Very slow improvement began again, and in about a year he was able to find his way about the streets.

Toxin of Bacillus Enteritidis of Gaertner.—E. P. Cathcart (*Jour. of Hygiene*, April, 1906, p. 112) reminds us that food infected with Bacillus botulinus or Bacillus enteritidis is much more likely to be dangerous on account of absence of obvious signs of putridity than that which is really putrid and has bad odor, color, and flavor. With Bacillus botulinus there is but faint odor; with Bacillus enteritidis no odor at all. The most dangerous of all is the latter, since cooking does not destroy its toxicity. Experiments concerning the toxins of various food poisoning organisms led Cathcart to the following (among other) conclusions: Cultures of Gaertner's bacillus grown in broth do not excrete any large amount of toxin into the fluid medium. Suspensions in broth made from ordinary agar cultures and killed by heat (60° C. thirty minutes) are fairly toxic. Gaertner toxin withstands heating to 100° C. for thirty minutes. Paratyphoid β bacillus cultures on antolysis yield a filtrate quite as toxic as those from Gaertner's bacillus. Paratyphoid toxin resists 100° for thirty minutes. Colon toxin is not heat resistant.

Boron Preservatives and Increased Mortality in England and Wales from Diseases of the Kidneys.—In reply to the dogmatic statement of H. W. Wiley, of the Bureau of Chemistry, that the large increase (asserted to be 30 per cent.) in kidney diseases in England is due without doubt to the use of coloring matters and preservatives in foods, and to the statement of Richard Jones that the gradual increase in the death rate from acute nephritis during the past twenty years is due to the excretion of food preservatives, especially the boron compounds, by the kidneys, B. FARADAY GILES (*Lancet*, 1906, ii, 1347) shows by a tabulation of the figures of the reports of the Registrar-general that before boron preservatives were in general use, the rate of increase in mortality from what were returned as nephritis and Bright's disease was even greater than afterward. Thus, during the decade 1861–1870 the annual rate per million (England and Wales) was 112; during 1871–1880 it was 199. Again, the annual mortality per million due to diseases of the urinary system, during the quinquennial periods 1861–65, 1866–70, 1871–75, 1876–80, ran thus: 246.4, 287, 326, 375. Hence before the use of boron preservatives had become important the mortality due to all diseases of the urinary system had already increased 52 per cent. in twenty years.

Poisoning by Saccharin.—LUTH, of Bajohren (*Lancet*, April 7, 1906), a town on the Russian frontier, describes the case of a woman who, after a dispute with her husband, had swallowed about 2 grams of saccharin. When a medical man arrived, about fifteen minutes later, he found her lying on the floor, and her condition was at first supposed to be due to alcoholic intoxication. She was unconscious, her face was flushed, she foamed at the mouth, and suffered from convulsive attacks with choking; her respiration was frequent and her pulse was weak, very quick, intermittent, and irregular. Under artificial respiration and massage of the heart, the pulse within half an hour became stronger and regular, and the respiration became normal. After about forty-five minutes the patient awoke and was quite well. Poisoning by saccharin is rather rare. It is remarkable that severe symptoms occurred within only a few minutes and as rapidly disappeared. In Germany saccharin is sold to the public only on medical prescription, and the woman had therefore purchased the drug from a neighboring Russian pharmacy.

Detection of Sterilized Milk.—Raw milk has the property of reducing certain substances such as indigo, litmus, and methylene blue, which are converted into colorless compounds, but whether this property is due to bacteria or to a natural reducing ferment is the subject of controversy. Schardinger found that methylene blue is decolorized more rapidly if a slight amount of formalin is added to the reagent, and on this fact H. Smidt (*Arch. f. Hyg.*, 1906, lviii, p. 313) bases his method of distinguishing raw from cooked milk, using as reagents an aqueous solution containing 2.5 per cent. each of formalin and saturated alcoholic solution of methylene blue, and another containing the same amount of methylene blue, but no formalin. Added in the proportion of 1 to 20 parts of milk, the first-named reagent alone is decolorized by fresh, raw milk, but both are decolorized by stale milk, while neither is affected by boiled milk in ten minutes, at a temperature of 104° to 122° F. From his experiments he concludes that if a simple solution of methylene blue is decolorized by raw milk, the action is attributable to bacteria present in the milk; that the speedy reduction of the methylene blue-formalin mixture by fresh, raw milk can be explained only by the presence of a ferment that acts catalytically on formalin; that the action of an added alkali on the milk sugar produces a reducing substance, and that prolonged boiling causes the production of substances which can decolorize methylene blue, but only at the boiling point. He believes that milk contains no preformed reductase.

The experiments of E. Seligmann (*Zeit. f. Hyg. und Infectionskrankheiten*, 1907, lii, 161) lead him to the conclusion that the active agents are bacteria. He found that while sterilized milk no longer can bring about the reduction, the property can be restored by inoculating it with a trace of sour milk. He obtained cultures of a short, thick bacillus, which on inoculation into sterile milk quickly restores the reducing property. He believes that reductase and superoxydase are organized ferments and are products of bacterial growth; and that certain decomposition products of casein have similar power to decompose hydrogen peroxide. The oxydases are not products of bacterial growth. C. J. Koning (*Revue gén. de laiterie*, 1906, No. 8, p. 186) asserts that milk contains no oxydases, and that positive reactions with tincture of guaiacum are due to the presence of peroxide in the reagent, but peroxydases are always present, and diastases as well. The latter can be produced in sterile milk by inoculation with the common milk bacteria, and they are increased in the milk of pathological udders. He asserts that Schardinger's reaction is due to reductase, which may be produced by bacteria, and, like catalase, is increased by udder disease and by the presence of leukocytes.

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ORIGINAL ARTICLES.

THE ETIOLOGY OF TUBERCULOSIS.[1]

INFECTION THROUGH FOOD AND CONTACT. THE ALIMENTARY CANAL AS A PORTAL OF ENTRY FOR THE TUBERCLE BACILLUS.

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Historical. The possibility of tuberculous infection through the digestive tract appears to have been first pointed out by Klenke, who, in 1846, gave the clinical histories of 16 children who had been nourished on cows' milk, all of whom showed tuberculous lesions of the intestines, glands, skin, or bones. While his observations lacked scientific exactness, he deserves credit for calling attention to a method of infection which recent research has shown to be of quite frequent occurrence.

Chauveau, in 1868, published the first experiments proving the possibility of tuberculous infection through the alimentary canal, employing for this purpose young cattle reared in a region where tuberculosis was practically unknown. Further successful experiments, carried out on a larger number of animals, were reported by him in 1872 and 1873, establishing at this early day, for cattle at least, the important role played by ingestion in the transmission of tuberculosis.

Villemin, in 1869, published confirmatory experiments carried out on rabbits and guinea-pigs. He was the first to use a tube for the introduction of the tuberculous material into the stomach.

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[1] An address delivered by invitation at the Fourteenth International Congress on Hygiene and Demography, Berlin, September 23–29, 1907.

The experiments of Gerlach, begun in Hanover in 1866, and later continued in Berlin on a much larger scale, gave the strongest support to the work of Chauveau. Gerlach was the first to experiment with the milk of tuberculous cows, and to prove that animals could be infected by the ingestion of such milk.

In 1870, E. Klebs reported experiments in which he had produced tuberculosis in animals by feeding tuberculous material from man as well as from animals, showing thereby the identity of the diseases. In 1873 he extended his experiments, employing tuberculous milk with positive results.

During the years 1870–73, Günther and Harms conducted at Hanover 94 feeding experiments, using animals of widely different species and tuberculous matter of various kinds. They obtained 24 positive and 70 negative or doubtful results.

Experiments with entirely negative results were reported by Colin, Semmer, and others. Those of Semmer were of especial importance. He fed upwards of 100 dogs on tuberculous material obtained from 30 cows, failing in every instance to produce tuberculosis. His results are at the present time interpreted as demonstrating the resistance shown by dogs to tuberculous infection.

Space does not permit me to go further into details, and I can only mention the admirable experiments of Viseur, Bollinger, Orth, Toussaint, Peuch, Baumgarten, Wesener, Leisering, Zürn, Saint-Cyr, Perroncito, Virchow and Schutz, Sydney Martin, Pearson, Schottelius, and others, all of which show that in animals infection takes place readily through the alimentary tract.

Many other facts, some experimental, some accidental, are at hand, confirmatory of the results above quoted. Among these may be mentioned the infection of large numbers of swine through feeding them milk from tuberculous cattle, one very striking instance of which has come under my personal observation in Pennsylvania. The spread of tuberculosis among swine and other animals through the use of skimmed milk and the waste products of creameries is also a well-established fact, and I believe that no one can seriously question the important role played by ingestion in the extension of the disease among the lower animals.

Many experiments have been carried out demonstrating the presence of the tubercle bacillus in various food products, such as milk, butter, etc., and the danger of such infected products for man has been generally recognized and accepted. In 1901, in his memorable London address, while discussing the relation of bovine tuberculosis to human health, Koch took the ground that infection through food could be assumed to have taken place with certainty only when the primary tuberculous lesion was located in the intestines. He claimed that this was rarely found to be the case, and argued that the danger of infection through food was slight.

The stand taken by Koch assumed that the tubercle bacillus was unable to gain entrance to the system through the intestinal wall without the production of a lesion at the point of entrance. In the discussion of infection through food, we must, however, take into consideration the possibility of infection through every portion of the alimentary tract with which the food comes in contact, beginning with the mouth. It has been thoroughly proved that the tubercle bacillus is able to penetrate the mucous membrane of practically the entire alimentary canal, as well as that of other parts of the body. Experiments demonstrating this will be quoted when considering the various portions of the alimentary tract which may serve as portals of entry.

MOUTH AND TONGUE, PALATE AND GUMS. Tuberculosis of these structures is rare, even as a secondary manifestation in advanced phthisis, when all the parts are constantly exposed to large quantities of sputum containing myriads of tubercle bacilli. Experimental infection is readily produced, followed by enlargement and caseation of the related glands, but as primary avenues of entrance they play a minor part in the spread of tuberculosis.

TONSILS. The tonsils have long been believed to serve as portals of entry, not only for the tubercle bacillus, but also for many other pathogenic microörganisms. They are not infrequently the seat of primary tuberculosis, and often show scars and other changes attributable to tuberculosis. In man they are so situated as to come into contact readily with food as it is swallowed. They are also exposed to the secretions of the nose and nasopharynx as they pass backward into the œsophagus; hence they are liable to infection from food as well as from material primarily lodging in the nares.

The literature concerning infection through the tonsils is extensive, and I cannot even mention much that is of great value and interest. In the examination of tonsils removed from otherwise healthy persons, Krückmann found primary tuberculosis in 2 of 25 cases; Gottstein, in 2 of 20; Pluder and Fischer, in 5 of 32; and Scheibner, in 4 of 60 cases. Wood has collected from the literature 1671 cases, 88 (5.2 per cent.) of which showed primary tuberculosis. These examinations were made by various methods, and probably fall short of the truth. Lartigau and Goodale have shown that systematic inoculation of animals gives a higher percentage of tuberculosis. In 75 cases Lartigau found 12 (or 16 per cent.) tuberculous. Dieulafoy, by the inoculation of guinea-pigs, found tuberculosis of the tonsils in 15 of 96 cases. His work has been criticised on the ground that he made no histological examinations, and was not careful to exclude the crypts, which may lodge tubercle bacilli. However, Latham, who avoided these sources of error, in 25 consecutive autopsies on children from three months to thirteen years of age, found 7 which were tuberculous—results practically identical with those of Dieulafoy. Confirmatory reports have been made by a large number of observers, many of which are included in the figures quoted from Wood. The susceptibility of the tonsils to tuberculous infection is shown by the examination of persons dead of phthisis.

Experimentally, the tonsils are readily infected, by direct application, as well as by feeding tuberculous material. In a series of experiments which I carried out at the laboratory of the State Live Stock Sanitary Board of Pennsylvania, swine fed with tubercle bacilli from man and from cattle developed generalized tuberculosis, with marked involvement of the tonsils, apparently primary, with necrosis and ulceration. Baumgarten observed, in his feeding experiments in 1884, that his animals showed primary tonsillar tuberculosis, with involvement of the related glands, and the same course was noted also by Orth in his studies. Woodhead traced tuberculous infection in swine from the tonsils into the neck, then into the thorax by the mediastinal and poststernal glands. Peira has shown that microörganisms are absorbed through the tonsillar epithelium, and, if virulent, may cause not only local disease, but be carried to the neighboring lymphatics, and thence to different parts of the body. Lexer confirmed these observations by a series of experiments on rabbits. Wood found that in swine the application of tubercle bacilli to the back of the mouth produced rapidly a primary tuberculosis of the tonsils, followed promptly by involvement of the submaxillary and cervical glands and extensive disease of the lungs. The mesenteric and bronchial glands were always diseased in about an equal degree. Aufrecht, by rubbing cheesy matter into the tonsillar region of rabbits, produced in one tuberculosis of the right tonsil and root of the tongue; in a second, tuberculosis of the cervical and mediastinal glands and the lung; in a third, disease of the mediastinal glands and the lung. All other organs remained free.

Grober concludes, from his thorough study of the question, that infection through the tonsil is the most frequent origin of apical tuberculosis. His experiments have demonstrated that from the cervical glands there is a direct route to the pleuræ and lungs, and that this leads especially to that portion of the lung most frequently the primary seat of tuberculosis—the apex. The evidence that the tonsils are frequently the portal of entry for the tubercle bacillus appears to me very conclusive, and it seems equally certain that food is the bearer of the infection in a large proportion of cases.

PHARYNX AND ŒSOPHAGUS. Tuberculosis of the pharynx and œsophagus is extremely rare, and, as a primary affection, is practically unknown. It is probable, however, that the intact mucous membrane of the pharynx at times allows the passage of tubercle bacilli, which first show their presence by enlargement of the related glands. Experimental evidence of this is strong, though it must be borne in mind that it is always difficult to place the exact point of entry in the absence of any lesion. In the case of a monkey, to which I fed tubercle bacilli enclosed in banana, and which died of pulmonary tuberculosis, the cervical glands were enlarged and caseous, though no lesion of the tonsils or neighboring parts could be found. It seemed probable that infection had taken place through some portion of the mouth or pharynx, though the tonsils may have been the point of entry.

STOMACH. There is no evidence that infection ever takes place through the stomach. Tuberculous disease of this organ is one of the rarest forms seen. The cause of this apparent immunity is not evident, but it is attributed to the protective action of the hydrochloric acid of the gastric juice. The action of the gastric juice on the tubercle bacillus has been the subject of considerable discussion and experiment. If this fluid had a germicidal effect on the tubercle bacillus, primary infection through the intestine would be manifestly impossible. It has been shown, however, that the tubercle bacillus is able to resist the action of the gastric juice for at least as long as the ordinary period of digestion.

INTESTINE. The intestine is very frequently involved in cases of phthisis, the autopsy reports of various pathologists giving figures that run from 30 per cent. to 90 per cent. In the great majority of these the lesion is unquestionably secondary, and due to swallowing sputum laden with tubercle bacilli. This does not, however, preclude the possibility that the pulmonary disease was caused in the first place by tubercle bacilli which gained entrance to the body through the intestine. In other words, the location of the primary lesion, on which so much stress has been laid, does not indicate with certainty the point of entrance of the invading organism. This appears to be especially true in children, in whom tuberculosis tends to become rapidly generalized.

The susceptibility to infection through the intestine unquestionably varies with the age of the individual. In young children the intestinal epithelium is looser, more delicate, and softer than in later life; the mucous membrane contains more fluid, and is more liable to slight injury (Furst). The intestinal catarrhs to which children are so liable also tend to lessen the natural resistance of the mucosa to invasion by microörganisms. Even before the discovery of the tubercle bacillus Weigert called attention to the permeability of the intestinal mucosa of young children to the virus of tuberculosis, stating that the mesenteric glands might be markedly swollen and cheesy, while the mucosa and lymph vessels showed no change whatever.

Tuberculosis of the mesenteric glands without lesion of the intestinal mucosa was observed also by Cohnheim, and since by von Bollinger, Wesener, Heller, Kossel, Aufrecht, von Hansemann, Rilliet and Barthez, Widerhofer, and many others. McFadyean and McConkey, by the inoculation of animals, demonstrated the presence of tubercle bacilli in 25 per cent. of children examined who showed no lesion of the intestine. Rabinowitsch also has shown that the mesenteric and bronchial glands of children, as well as of adults, may contain living tubercle bacilli, though there had been no clinical sign of disease and the microscope gave negative results. In his feeding experiments, Orth produced tuberculosis of the mesenteric glands without change of the intestine. Feeding with perlsucht material caused tuberculosis of the various organs without perceptible lesion of the intestine or peritoneum.

PRIMARY INTESTINAL TUBERCULOSIS. Pathologists give the most contradictory reports as to the frequency of primary intestinal tuberculosis. It is impossible to explain satisfactorily the widely differing findings, coming, as they do in some instances, from hospitals in the same city. They are accounted for in part by differences in the methods of examination and interpretation of findings; in part, by the class of patients frequenting certain hospitals; and in part by a real difference in the incidence of intestinal infection in various communities, brought about by local customs and habits. In all reports a large percentage of cases is found in which it has been impossible to determine the site of the primary lesion, and from many of them it is difficult to draw correct conclusions, since they have been compiled to demonstrate certain points, and do not give clear details as to other important features.

From England only do we get statistics which approach uniformity. English pathologists who have studied tuberculosis in children are practically unanimous in believing that infection through the intestine is of frequent occurrence. Still, Shennan, Symes and Fisher, Guthrie, Carr, Ashby, Batten, and Kingsford report on 1560 autopsies on children in which the primary lesion was found in the intestine 290 times, or in 18.6 per cent. of all cases. These were seen in different parts of England, though the largest number came from London. Woodhead, in 127 autopsies on children, found the mesenteric glands involved 100 times; in 69 the bronchial glands were also involved; in 27 the bronchial glands alone, and in 14 the mesenteric alone.

In the United States of America, Northrup, Holt, and Bovaird report on 369 cases, all from New York or its environs, with only 5 of intestinal origin—a little more than 1.3 per cent. Holt contributed 119 of these autopsies, in none of which did he consider the intestine the seat of the primary lesion. It is interesting to note, however, that he found the mesenteric nodes involved in 35 per cent., and the intestines in 37 per cent. of these cases. From the Children's Hospital of Philadelphia, Hand reports 115 autopsies, with 10 cases (8.7 per cent.) of primary intestinal localization, and 1 of tonsillar invasion. In 29 cases the point of invasion could not be determined.

In this connection the work of Councilman, Mallory, and Pearce is most instructive. They found tuberculosis in 35 of 220 children dead of diphtheria. In 18 of these the mesenteric lymph nodes were tuberculous, with involvement of the intestine 6 times; and in 7 cases the mesenteric glands were diseased without involvement of any other part of the body. In 13 cases (37.1 per cent.) the infection was evidently through the digestive tract.

The statistics from Germany are most contradictory and confusing. As is well known, Koch considers primary intestinal tuberculosis very rare, having collected only some 28 cases. Before accepting a case as being due to food, Koch demands a train of evidence, as follows: Clinical and anatomical data of the disease and its point of invasion; the exclusion of all other sources of infection; the coincident infection of others using the same milk; demonstration of udder tuberculosis in the cow furnishing the milk. It is needless to say that such a train of proof is practically impossible to obtain. Furthermore, neither Koch nor anyone else asks for such evidence in determining the source of respiratory tuberculosis—or, in fact, of any other disease.

Baginsky, in a series of 806 autopsies, 144 of which showed tuberculosis, found only 6 in which he considered the intestinal lesion the oldest. In a second series of 933 autopsies on children he did not once find intestinal tuberculosis without involvement of the lungs and bronchial glands. This statement does not appear to me to be of much value, since no indication of the site of the primary lesion is given. Biedert found only 16 cases of primary intestinal tuberculosis in 3104 autopsies on children. Widerhofer, Heubner, Benda, von Hansemann, Ganghofner, and others agree in regarding primary intestinal tuberculosis as comparatively infrequent. Von Hansemann saw only 25 cases in seven years. Widerhofer, in an analysis of 418 cases of tuberculosis in children, found involvement of the intestine (not primary) 101 times. Of these 43 (42.5 per cent.) were between the ages of two and five years, when food infection is most liable to occur.

On the other hand, we have a mass of testimony from equally competent observers which agrees closely with the figures from England. Hueppe, while giving no statistics, says: "The number of cases (primary intestinal infection) may fairly be reckoned as between 25 and 35 per cent. of all deaths in children from tuberculosis." Hof, in a systematic study of the autopsy records of the Pathological Institute at Kiel, found 2697 cases of tuberculosis in adults, 159 (5.9 per cent) of which were primary in the intestine; while in 84.9 per cent. the respiratory tract was primarily involved. In children there were 936 cases of tuberculosis, 235 (25.1 per cent.) of which showed evidence of infection through the intestine, and 527 (56.2 per cent.) respiratory infection.

Wagener, in 600 autopsies, 76 of which were on children, found primary intestinal tuberculosis in 16 to 21 per cent. Heller, among 714 fatal cases of diphtheria, found tuberculosis 140 times, in 53

(37.8 per cent.) of which the origin was primary in the intestine. In a later series of autopsies Heller found intestinal tuberculosis in 12 per cent. of the adults and 26 per cent. of the children. Nebelthau, in 26 autopsies on tuberculous children at the Halle Polyclinic, found the infection was primary in the intestine in 5 (19.2 per cent.), and in the respiratory tract in 9 (34.6 per cent.), while in 12 (46.1 per cent.) both tracts were infected. Kossel, in 14 children dead of other diseases, found tuberculosis of the bronchial glands 10 times, and of the mesenteric glands 4 times. In 22 children who died of tuberculosis, only once did he find the disease confined to the intestine. Lubarsch, in 297 autopsies on children, found tuberculosis in 63, of which 14 (21.2 per cent.) were primary in the alimentary tract.

The marked differences in the findings of pathologists even in the same city has been mentioned. In Berlin, von Wagener, at the Bethanien Hospital, from October, 1903, to October, 1904, found in 67 autopsies on children from one to fifteen years old, primary intestinal tuberculosis in 16 per cent., while at the Charité, from October, 1902, to December, 1903, Orth found in 131 children only 1.5 per cent. of undoubted primary intestinal and mesenteric disease. Edens, among 31 tuberculous children at the Bethanien Hospital, seen from October, 1904, to October, 1905, found 35.5 per cent. of primary intestinal disease, while Orth during the same years found only 8 per cent. in 77 children (Rabinowitsch). At the same hospital, from October, 1905, to October, 1906, Edens found 18 cases of primary intestinal tuberculosis in 409 autopsies, at all ages. Of the 74 children from one to fifteen years old, 21 of whom were tuberculous, 10 (47.6 per cent.) showed primary intestinal involvement. Of 319 cases from fifteen to ninety years old, 130 were tuberculous, and 8 (6.2 per cent.) showed primary intestinal involvement.

In Copenhagen, Febiger found 13 cases of tuberculosis beginning in the intestine among 213 autopsies on persons of all ages, or 6 per cent. for all cases and 11 per cent. for tuberculous cases. Ipsen carried on the work, and gives us a series of 600 autopsies, including those of Febiger. In 32 cases primary tuberculosis of the digestive canal was found, which gives 5.3 per cent. for all cases and about 10 per cent. for tuberculous ones. These figures include 102 children from birth to fifteen years of age who died from various diseases. Among these 6 showed tuberculosis limited to the intestine.

Harbitz gives the following results obtained from the examination of 117 cases: Primary in the respiratory tract, 48 (41 per cent.); primary in the digestive tract, 26 (22 per cent.); primary in the digestive or the respiratory tract, 24 (20.5 per cent.); general lymph-node tuberculosis, 11 (9.4 per cent.); doubtful, or other primary seats, 8 (6.8 per cent.).

Studies on the bodies of children who have died of diseases other than tuberculosis, such as those reported by Councilman, Mallory, and Pearce, Heller, and Kossel, are particularly valuable in the determination of the avenue of entry for the tubercle bacillus. The disease tends to become generalized so rapidly in children that at autopsy it is often impossible to tell by what route infection occurred. When death has been due to other causes the tuberculous lesion is usually localized, and often confined to the glands in relation to the point of invasion; hence the portal of entry can be determined with great certainty.

Statistics of Primary Intestinal Tuberculosis. Furst has collected the reports of 124 cases of undoubted primary intestinal tuberculosis, 30 almost certainly primary, and 23 doubtful ones. He concludes that there are on record some 160 cases of primary intestinal involvement which cannot be questioned. To these we may add 18 recently reported by Edens, and about 20 of which I have knowledge, including the cases published by Northrup, Holt, Bovaird, Hand, Ravenel and Fife, and some unpublished ones. Furst leaves out of consideration entirely the 290 cases given by the English pathologists quoted above.

The total, however, is not large when we consider the enormous death rate from tuberculosis. Numerous experiments long ago convinced me that an infection through the alimentary canal often shows itself first in the thorax; and I believe it is unfair to consider as cases of intestinal infection only those in which the primary lesion is found in the intestine or mesenteric glands. The question hinges largely on the ability of the tubercle bacillus to pass through the intestinal wall without causing a lesion at the point of entrance, and this will be next considered.

Permeability of the Intestinal Wall. It is believed by many pathologists that the tubercle bacillus is able to penetrate the normal mucous membrane of the intestine without leaving any lesion at the point of entrance. In fact, this seems to be true of the mucous membrane of other parts of the body also, as shown by the great frequency with which the various groups of glands are found to be tuberculous with no demonstrable lesion of the surfaces they drain. In 1000 swine, infected through food, Ostertag found the glands of the throat, neck, and mesentery tuberculous, while the mucous membrane of the intestine was always free from disease. The passage of tubercle bacilli through the intestine appears to be most likely to occur during the digestion of fats, as shown in the experiments given below.

Von Baumgarten has recently denied that tubercle bacilli penetrate the body membranes without causing a lesion at the point of entry. In the numerous experiments by himself and his students he has found that animals fed with milk containing tubercle bacilli developed an intestinal tuberculosis with general infection. If the infection was rapidly fatal the intestinal lesions were very small, being found only in sections, but widespread. The mesenteric glands showed more advanced disease and, apparently, of longer duration. In aspiration tuberculosis also he has not been able to discover disease of the lymph glands without involvement of the trachea or lungs. Von Hansemann accounts for cases of abdominal infection without intestinal lesion on the ground that a slight tuberculosis of the intestine may disappear, leaving no sign of its location. The truth of this observation must be admitted, but it does not explain the numerous experiments in which the tubercle bacillus, as well as other organisms, have been shown to penetrate the intestinal wall in the course of a few hours even when there is no demonstrable lesion.

Orth, in 1879, and Cornet, in 1880, observed bacilli pass step by step through the fully developed mucous membrane of the uninjured intestine and reach the lymph channels and mesenteric glands, leaving no recognizable trace behind (Furst). Dobroklonski, in 1890, working under Cornil, showed that the tubercle bacillus would quickly penetrate the healthy wall of the intestine in guinea-pigs. Desoubry and Porcher, students of Nocard, showed in dogs that during the digestion of fats large numbers of bacteria were carried through the intestinal wall and could be detected in the chyle within a few hours after the meal was given. If food free from fat was given few or even no bacteria could be found. Nicolas and Descos, in 1902, found that tubercle bacilli given to dogs in fatty food reached the chyle within three hours. During feeding experiments conducted at the laboratory of the State Live Stock Sanitary Board of Pennsylvania, we frequently observed extensive tuberculosis of the lungs and thoracic glands in animals which showed slight or even no involvement of the intestine. In 1902-3, I introduced into the stomachs of a number of dogs tubercle bacilli suspended in an emulsion of melted butter and warm water, using a tube in order to prevent possible infection through the trachea. The dogs were killed after three and one-half to four hours, during active digestion, as much chyle as possible was collected, and the mesenteric glands were removed. Guinea-pigs were inoculated with this material. Tubercle bacilli were demonstrated in abundance in 8 of 10 experiments. The dogs were kept on soft food for some days before the experiments, and were purged with castor oil, in order to rid the intestine of all foreign matter which might injure the mucous membrane. Numerous sections of the gut were examined also, but no injury could be detected. Römer and von Behring have shown that the tubercle bacillus, and even the anthrax bacillus, which is very much larger, passes readily through the intestinal mucosa of young guinea-pigs. A single feeding with a minute dose of tubercle bacilli frequently produced infection, the glands of the neck always being involved, and later there frequently developed a type of disease usually regarded as the expression of an inhalation tuberculosis. As is well known, von Behring believes that the origin of epidemiological tuberculosis in man and epizoötic pulmonary tuberculosis in cattle is a primary intestinal infection taking place in early life.

The subject has been recently studied by Calmette and his fellow-workers Guérin, Vansteenberghe, and Grysez. In numerous experiments they have found it impossible to produce anthracosis of the lungs even when animals were compelled to breathe an atmosphere saturated with lamp-black, provided the œsophagus was closed. On the other hand, when lamp-black was introduced into the stomach by means of a tube, or mixed with food, anthracosis of the lungs appeared rapidly. When tubercle bacilli, dry or moist, were administered by inhalation, by intratracheal insufflation, or by direct inoculation into the trachea, the bacilli never penetrated farther than the first branches of the bronchi. The introduction of tubercle bacilli into the stomach through a tube, in order to avoid danger of respiratory infection, always produced tuberculosis rapidly. They confirm the observation that tubercle bacilli readily penetrate the intestinal wall without leaving a lesion. They have traced the bacilli, and find that as soon as they reach the chyle vessels they are taken up by the leukocytes, which, from this time, act as carriers and convey them to the related glands, where they are retained for a longer or shorter time, reaching finally the thoracic duct, and through it the circulation. In young animals the mesenteric glands retain the leukocytes with their englobed bacilli for some time, the glands enlarging in proportion to the intensity of the infection, the lungs becoming involved secondarily. In the adult the glands do not retain the bacilli nearly so long, and they can be detected in the lungs twenty-four hours after their introduction into the stomach. Calmette concludes that pulmonary tuberculosis acquired at any age may be due to recent intestinal infection.

Besanti and Panisset have shown that tubercle bacilli given to dogs suspended in soup reach the blood of the heart within four to five hours. In 4 out of 6 experiments they obtained positive results. Bartel found that after a single dose tubercle bacilli penetrated the uninjured intestine and reached the mesenteric glands during the following digestive period. Von Weismayr, as the result of his studies, is of the opinion that microörganisms can penetrate the intact intestinal wall, and be carried by the lymph cells through the lymph channels to the related glands. Schlossman and Engel have shown that when tubercle bacilli suspended in milk or cream are injected into the stomachs of young guinea-pigs through an incision in the abdominal wall they reach the lungs within six hours, as shown by killing the animal and inoculating portions of the lungs into other pigs.

Infection by Bovine Tubercle Bacilli. In 1901, I isolated from the mesenteric glands of a child who died of tuberculous meningitis following primary intestinal infection, a culture (B B) which, after the first generation, showed all the characteristics of the bovine bacillus, killing cattle rapidly. A second culture (U) also from a case of meningitis, though of doubtful origin, was of the human type in growth and morphology, but showed high virulence for dogs and calves. A third culture (K K), reported by Dr. Fife and myself from a case of primary intestinal tuberculosis, was bovine in all respects, including virulence for calves. In America, de Schweinitz has isolated bacilli of the bovine type from children three times; Theobald Smith, once; and Mohler, once. In Europe, Febiger and Jensen, Wolff, Westenhoffer, the Commission of the Imperial Board of Health, and the English Royal Commission have isolated from cases of human tuberculosis bacilli of the bovine type, fully virulent for cattle. The German Commission examined 56 cultures from man, and found 6 (10 per cent.) to be bovine. These were, with one exception, from children with mesenteric or intestinal lesions. The English Commission studied 60 cultures isolated from man, of which 14 (23 per cent.) proved to be of the bovine type; 28 of the cases from which these cultures were obtained had histories pointing to intestinal infection, and of these 13 (46.4 per cent.) gave cultures of the bovine type.

The opportunities for the inhalation of the bovine tubercle bacilli by human beings are slight, except, perhaps, for persons who habitually care for cattle, and, compared to those for ingestion, are insignificant. There is practically no possibility for the inhalation of bovine bacilli by children even if we admit that the aspiration of particles of food may occur. We are, therefore, justified in concluding that in those cases from which the bovine bacillus has been isolated the infection has taken place through the digestive tract.

INFECTION THROUGH CONTACT. It is impossible to state how frequently contact is concerned in the spread of tuberculosis. In the great majority of cases of infection by this means the source is never suspected. Contact plays an important part in the spread of many other diseases, and is probably more frequently the mode of infection in tuberculosis than we believe. Kissing of infants by tuberculous parents is especially dangerous, as is also the habit, common among mothers and nurses, of tasting the infant's food in order to test its heat. Demme recounts the case of a tuberculous nurse who fed her charges with a spoon she constantly put into her own mouth, with the result that four died with primary isolated intestinal tuberculosis.

The hands of consumptives are frequently contaminated with sputum and excretions. Baldwin showed, by the inoculation of guinea-pigs with the washings from the hands of 10 private patients, that 8 were the bearers of living tubercle bacilli. Of 5 sanatorium inmates, only 2 gave positive results. Children playing on floors soiled with sputum may readily get their hands contaminated, and thus convey bacilli to their mouths. The hands also become contaminated by means of handkerchiefs, rags, sputum cups, clothing, bedding, etc., used by consumptives. Local infections have been observed through the use of towels, handkerchiefs, and other articles, and in the laundering of such articles. Wounds or abrasions on the hands, face, and other parts of the body are liable to become infected through soiled bandages or tuberculous attendants, as in cases reported by Verneuil, Tuffier, Kraske, Wahl, and others. Elsenberg observed infection of the penis after circumcision by a tuberculous priest, who used his mouth to arrest bleeding. Similar observations are recorded by Meyer, von Lehman, and Hofwokl.

Inoculation of the hands through wounds is often seen, chiefly among physicians and veterinarians engaged in postmortem work on man and animals. It occurs also in butchers and others handling tuberculous carcasses. The usual result of such inoculation is the anatomical tubercle, though tuberculosis verrucosa cutis sometimes follows, especially in those handling animal products. Such cases have been reported by Gerber, Lassar, Spronk and Höfnagel, Pfeiffer, Tscherning, Krause, Joseph and Trautman, Schutz, Kliene, de Jong, Mueller, Hartzell, Ravenel, and many others. In the great majority of such cases the infection remains more or less local, and the health does not suffer. In some it becomes generalized and death follows, as in the cases of Pfeiffer and Hartzell. The death of Wally, principal of the Royal Veterinary College of Edinburgh, was attributed to such an infection from a bovine source, and it is well known that Laennec believed that his phthisis originated through a postmortem wound. The proof in none of these cases is entirely conclusive. Among other modes of infection through contact which have been reported may be mentioned the bites of consumptives (Verchère, Jeanselme); vaccination; skin transplantation (Czerny); rubbing with phthisical saliva during tattooing (Jadasohn); the use of milk as a soothing application to injuries, etc. Such methods are exceptional, and are of little importance in spreading the disease.

Sources of Infection. The scope of this paper does not permit me to discuss this topic at length. It has been repeatedly proved that the tubercle bacillus passes into the milk of tuberculous cows even when there is no disease of the udder (Smith, Rabinowitsch and Kempner, Ravenel, and others), and, further, that tuberculosis is communicable from animals to man. The chief source of infection through food, strictly speaking, must then be considered the milk and other products of tuberculous cattle. However, food may become contaminated by sputum and excretions from consumptives. Our honored president has clearly demonstrated the danger from droplets expelled during coughing, sneezing, etc. Acting on the suggestion contained in his work, I have shown that cows throw out many tubercle bacilli in the same way. These floating particles, as well as the dust of dried sputum, are drawn into the nose and mouth during inspiration, and pass into the œsophagus with the secretions which are constantly swallowed, thus gaining access to the alimentary canal.

Conclusions. 1. The alimentary tract is a frequent portal of entry for the tubercle bacillus.

2. The tubercle bacillus is able to pass through the intact mucous membrane of the alimentary tract without producing a lesion at the point of entrance. This takes place most readily during the digestion of fats.

3. The bacilli pass with the chyle through the lacteals and thoracic duct into the blood, which conveys them to the lungs, where they are retained largely by the filtering action of the tissues.

4. Infection through the alimentary tract is especially frequent in children.

5. Milk from tuberculous cows is the source of infection in many cases. Our present knowledge does not enable us to state the exact proportion of cases of tuberculosis due to this cause, but it is probably considerable.

6. Tuberculosis can be communicated by contact, such as kissing, soiled hands, accidental injuries in postmortem work, or during the cleansing of vessels used by consumptives, etc. These modes of infection play a comparatively small part in the dissemination of the disease.

THE ETIOLOGY OF CERTAIN CASES OF LEFT-SIDED INTRA-ABDOMINAL SUPPURATION—ACUTE DIVERTICULITIS.[1]

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During the past ten years I have observed six cases of acute intra-abdominal abscesses, all originating in the left lower quadrant and presenting symptoms so similar to acute appendicitis as to lead me to consider the possibility of transposition of the viscera.

The first of these cases occurred at the New York City Hospital. A middle-aged woman was admitted to the surgical ward, suffering from pain, tenderness, and muscular rigidity in the left inguinal region. The history of the onset of the symptoms was difficult to obtain, but as she regarded the condition as one of acute indigestion, it is probable that it was sudden in its onset, and in the beginning it was associated with nausea and vomiting. When first seen by me there was moderate fever, some acceleration of the pulse, and, on careful palpation, a well-defined mass could be felt occupying a position midway between the left anterior superior spinous process and the median line. Operation revealed a well walled-off intraperitoneal abscess, containing about 100 c.c. of foul-smelling pus. There was no escape of gas, and no evidence of a fistulous opening; in fact the intestines were so matted together with fibrinous exudate that they could not be recognized, and no effort was made to separate them. The wound was closed with drainage, and she made a rapid and satisfactory recovery.

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[1] Read at a meeting of the American Surgical Association, Washington, D. C., May 9, 1907.

My second case was also seen at the New York City Hospital. An elderly woman was admitted suffering from pain, tenderness, muscular rigidity, and a mass in the left inguinal region. These symptoms had been present for several days. On admission, she had a slight elevation of temperature and looked moderately septic. The indurated area in this case was situated somewhat nearer the iliac spine, but in all other respects it clearly resembled the case just reported. On operation the peritoneal cavity was apparently not opened, the incision, which was quite near Poupart's ligament, leading directly to a large abscess cavity, which contained perhaps 250 c.c. of thick pus. The cavity, which was freely explored through a large incision, was seen to extend inward, beneath the descending colon and upper part of the sigmoid, the under surface of the latter apparently forming a part of the roof of the cavity. The bowel wall at this point was covered with a thick, fibrinous exudate, and it was thought by several who saw it that there might have been a small perforation, which had been closed by the exudate. After evacuating the pus the wound was partly closed and the cavity packed with sterile gauze. She made a slow but satisfactory recovery.

In both of these cases my records are imperfect, and I am obliged to rely largely upon my memory, but I distinctly recall that the cases made a strong impression upon my mind, and that I discussed their possible etiology with several of my colleagues. In the beginning I was strongly in favor of the belief that we had to do with a transposition of the viscera and a left-sided appendicitis. In my second case, however, this theory was easily disproved, as we had a retroperitoneal abscess, which, if it had been caused by a perforated appendix, would certainly have shown some trace of the organ by our careful inspection. My final conclusion was that in all probability the infection took place through some very small perforation of the sigmoid, as from a fish bone or other pointed foreign body, or possibly through a small area of necrosis occurring in a dysenteric or tuberculous ulcer.

My third case was observed three or four years later, at the Roosevelt Hospital. A man, sixty years of age, was admitted to the hospital with a diagnosis of abdominal tumor. On questioning him we elicited the fact that he had always enjoyed fair health, but had been constipated for a number of years. Four weeks before his admission he had complained of more or less severe abdominal pain, which had prostrated him and compelled him to take to his bed. This continued for several days, and, he thought, was associated with some fever. As the pain and fever subsided he noticed a painful swelling in the left inguinal region, which gradually increased in size until his admission to the hospital, when it was as large as a small cocoanut.

On examination, there was practically no muscular rigidity. Inspection revealed an oval mass midway between the umbilicus and the anterior spine of the ilium on the left side. This mass was flat on percussion, somewhat movable, smooth on the surface, of the density of an ovarian cyst, and was elastic but not frankly fluctuating. The tenderness to pressure was so slight as strongly to suggest a new-growth rather than an inflammatory mass. This view was also strengthened by the absence of fever.

Under ether anesthesia an exploratory incision was made through the left rectus muscle. The subperitoneal fatty tissue was œdematous. On dividing the peritoneum the incision opened into a large abscess cavity, which contained about 500 c.c. of creamy, odorless pus. The wall of the cavity was made up of omentum and several loops of intestine, all matted together with a dense, fibrinous exudate. The cavity of the abscess was washed out, the wound partly closed by layer sutures, and a large drain inserted at the upper angle. He made a satisfactory convalescence.

This case, while it cast no additional light on the etiology of this condition, served to reawaken my interest in the subject. About this time, in discussing the subject with my colleague, Dr. Evan Evans, he suggested as a possible cause, the rupture of a small acquired diverticulum of the sigmoid or descending colon. These diverticula are of fairly frequent occurrence in elderly people, are generally multiple, and, on account of their thin walls, might easily rupture if inflamed or distended by a foreign body. I, therefore, made a systematic inquiry of a number of pathologists of my acquaintance, who were making large numbers of autopsies; and, while most of them had seen these diverticula, occasionally present in large numbers, none could recall an instance in which rupture had occurred as a result of pressure of a foreign body or an inflammatory process.

Intestinal diverticula, as is well known, are divided into two general classes—the congenital and the acquired. The most frequently observed type of the congenital variety is the one known as Meckel's diverticulum. As recently described by Léon Cahier,[2] in his admirable essay upon the subject, a Meckel's diverticulum possesses the following characteristics: It is single; it has a rectangular implantation into the free border of the terminal portion of the ileum, generally in the neighborhood of the ileocecal valve; it is made up of all of the coats of the intestine; it is generally more than 2 cm. in length; and it has a terminal filament which may be free or attached to the abdominal wall, the mesentery, or another part of the intestine. The acquired diverticula, on the other hand, are, as a rule, multiple, small, thin-walled, and round or ovoid in shape; they may be found in any part of the intestinal canal, but are more frequent in the left colon and the rectum, and are in reality hernial protrusions of the mucous membrane through the separated fibers of the muscular coat.

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[2] L'inflammation des diverticules intestineaux ou diverticulite, Revue de chirurgie, September, 1906.

While congenital diverticula have been known since the early part of the eighteenth century to be the not infrequent cause of intestinal strangulation, and during the last fifty years to be the occasional cause of peritonitis from perforation, the pathological importance of the acquired diverticula in the production of intra-abdominal abscess has been practically ignored by clinicians, and barely mentioned by pathologists. Wallmann,[3] in 1858, reported 9 cases of acquired diverticula of the colon, of which 1 was in the ascending portion, 3 in the transverse, 3 in the descending, and 2 in the sigmoid. In no instance, however, was any other pathological lesion present. Leube[4] has described these diverticula as hernias of the mucous membrane, and stated that they occur most frequently along the mesenteric border of the bowel. Klebs called attention to the relationship between these protrusions and the bloodvessels of the bowel. In 1900 Hodenpyl[5] exhibited to the New York Pathological Society two specimens of acquired diverticula, one of the duodenum and one of the colon. The latter (see figure) presented seventy-nine distinct pouches, the majority being in the lower segment of the gut. He expressed the opinion that they were probably due to internal pressure caused by chronic constipation. Other interesting communications on the subject have been made by Max Edel[6] and Graser.[7]

Regarding the pathological importance of these diverticula, I have been able to glean very little from the literature. Sydney Jones,[8] in 1858, was, perhaps, the first to mention it. He described a case of intestinal-bladder fistula, which, he believed to be due to ulceration of a sigmoid diverticulum. Loomis,[9] in 1877, described a case of peritonitis due to inflammation of multiple diverticula of the left colon, several of which contained concretions. Birch-Hirschfeld,[10] in 1887, called attention to the possibility of diverticulitis with perforation and the formation of intra- or extra-peritoneal abscess. George Biggs,[11] in 1894, reported to the New York Pathological Society a specimen showing multiple diverticuli of the sigmoid, one of which had perforated and caused an extraperitoneal abscess. More recently Fischer[12] has reviewed the entire subject, described four specimens, and expressed some doubt regarding the role of constipation as a causative factor. The most satisfactory and comprehensive exposition of both the pathological and clinical aspects of the subject is the paper by Edwin Beer, in the AMER. JOUR. MED. SCI., July, 1904, p. 135.

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[3] Virch. Arch., 1858, xiv.

[4] Ziemssen's Spec. Path. und Therapie.

[5] Trans. New York Path. Soc., March 29, 1900.

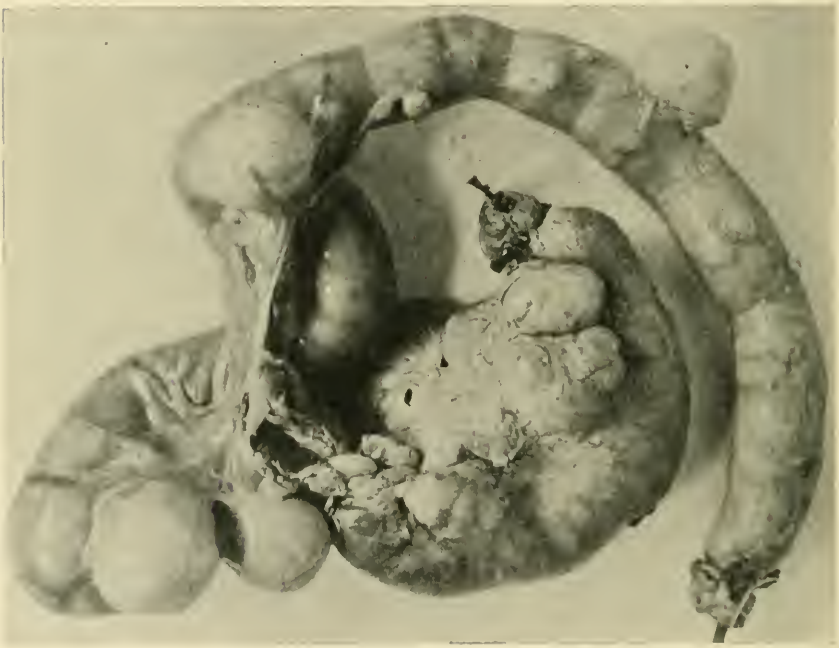
[6] Virch. Arch., 1894, cxxxviii.

[7] Münch. med. Woch., 1899, Nr. 22, and Arch. f. klin. Chir., 1899.

[8] Trans. Path. Soc. London, 1858.

[9] Trans. New York Path. Soc., 1877.

[10] Path. Anat., 1887.



Hodenpyl's specimen of multiple acquired diverticula of the colon (sigmoid flexure).

It will thus be seen that from time to time during the past half century pathologists have observed and reported specimens showing inflammation, necrosis, and perforation, with or without the presence of fecal concretions, of these false diverticula of the left colon, and have clearly demonstrated the relationship between these lesions and a general or localized peritonitis. For the most part, however, these communications have been buried in the transactions of pathological societies or have appeared in periodicals not largely read by clinicians. This circumstance and the rarity of the condition will easily account for the fact that the disease is not described in our clinical text-books, nor generally recognized by the profession.

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[11] New York Path. Soc., March 28, 1894.

[12] Jour. Exp. Med., 1900, p. 333.

I have long been impressed with the probable relationship between these diverticula and left-sided abdominal abscess, but have been unable to verify this impression until quite recently. The following case, while it does not prove the relationship absolutely, presents features which render it highly probable:

F. M., aged forty-seven years, was seen by me in consultation in August, 1906. The patient for twenty years had been a high liver and a hard drinker. One year before, he had had an attack of acute nephritis with uremia, from which he had partially recovered. Six weeks before being seen by me he had experienced an attack of abdominal pain, with nausea and vomiting. The pain was at first located in the centre of the abdomen, but later shifted to the left lower quadrant. There was moderate fever and considerable prostration during the first few days following the onset of the symptoms. Later, a tender swelling was noticed just to the left of the median line, and below the umbilicus. This mass slowly increased in size, and at times would be so painful as to interfere with sleep. The symptoms were only of moderate severity, however, and he remained under medical treatment for several weeks in the country, until increased fever, chilly sensations, and augmented tenderness in and about the mass led his medical adviser to advocate his removal to a hospital. He was accordingly brought to New York and placed under the care of my colleague, Dr. E. B. Shelby, with whom I saw him in consultation on the following day.

At my first examination I found the patient with a temperature of 100°, pulse 110. He complained of no discomfort except when moved about or palpated. On inspection, a swelling could be distinctly seen below and to the left of the navel. On palpation there was slight rigidity of the left rectus muscle, and an oval mass about the size of an egg-plant could be felt, which was distinctly tender to pressure. The appendix region was free from tenderness. The free border of the liver could be felt well below the costal arches; the spleen was large to percussion, but could not be palpated. The blood showed a moderate leukocytosis, with an increased percentage of polynuclear leukocytes. The heart and lungs were negative. The urine showed a trace of albumin with casts, but was sufficient in quantity.

He was removed to the Roosevelt Hospital, where, under general anesthesia, an incision was made through the outer border of the left rectus muscle, which exposed a large, well walled-off abscess, containing gas and about 500 c.c. of foul-smelling pus. On account of the poor physical condition of the patient, no effort was made to break up adhesions, with the view to determining the point of intestinal leakage.

He rallied well from the operation and made a tardy convalescence. The wound healed very slowly; one small sinus persisted and led downward toward the pelvis, and through it small quantities of gas would occasionally escape. On three or four occasions during his convalescence secondary abscesses formed in the neighborhood of the sinus. These, as a rule, discharged spontaneously. He was up and about, however, and after October 1 was able to go down town and attend to his business.

Later, as his health improved, an attempt was made to find and close the intestinal opening. On December 5, under ether anesthesia, after evacuating and packing an abscess of the abdominal wall, the sinus was followed down to the region of the sigmoid flexure in the pelvis, without opening the peritoneal cavity. A second incision was made to the right of the median line and the peritoneal cavity was opened and explored. The intestines were generally free from adhesions. Two portions of the gut, however, were firmly adherent to the wall of the abscess; the first, which in all probability was the primary lesion, was situated about the middle of the sigmoid; the second, probably a secondary communication, was a loop of small intestine adherent to the upper part of the abscess wall above and to the left of the umbilicus. Both of these adhesions were separated, and the minute openings closed with purse-string sutures. The right abdominal wound was united by layer sutures; the wound on the left side exposing the abscess cavity was partly united and drained with gauze. The patient bore the operation well, but vomited a good deal after coming out from the ether. There was practically no reaction during the first twenty-four hours, the temperature and pulse remaining at or near the normal. The vomiting, however, was troublesome, and on the second day after operation he vomited a large quantity of dark fluid, with clotted blood, which greatly weakened him, and was followed by a rapid rise in pulse and temperature. The vomiting was repeated several times, and eventually resulted in his death. At no time was there any local sign of peritonitis. We attributed the gastric hemorrhage to cirrhosis of the liver.

In my next case the relationship between an acute diverticulitis and an intra-abdominal abscess is clearly proved:

S. D., aged forty-five years, had always enjoyed good health. He had never suffered from digestive disturbances, suggesting appendicitis, gallstone colic, or peritonitis. In August, 1902, while at dinner, he was suddenly seized with an attack of abdominal pain, nausea, and faintness, which necessitated his leaving the table and returning to his room. The attack soon passed off and he was able to join his friends later in the evening. The following night proved a restless one, as he had more or less constant pain in the lower portion of the abdomen, which prevented sleep, and at times was accompanied by nausea and general bodily weakness. The following day he continued to feel badly, but kept up and about for the reason that he was a guest at a country house, and did not wish to inconvenience his host. Later in the day he went for a drive, and suffered acutely from the jolting of the vehicle. In the evening he was obliged to call a physician, who, after an examination, pronounced the case one of colitis. He returned to the city the following day, and as the symptoms continued, he remained in bed. During five days he continued to suffer with pain in the lower left quadrant of the abdomen, fever, and general malaise.

When first seen by me the temperature was 103°; pulse, 110; leukocytes, 17,000. There was marked rigidity of the left rectus muscle and a tender mass in the iliac fossa.

He was immediately removed to the Roosevelt Hospital, and, under ether anesthesia, an incision was made over the most prominent portion of the tumor. After dividing the tissues of the abdominal wall, a large abscess cavity was entered, which contained about 120 c.c. of foul pus, and an oblong, fecal concretion. On washing out the abscess cavity, a small ulceration was seen in the wall of the sigmoid, through which escaped a small amount of fecal matter. The cavity was packed with sterile gauze, the wound partly united, and a dressing applied.

After operation the temperature and pulse rapidly declined to the normal, the pain ceased, and the appetite returned. The discharge from the abscess cavity gradually diminished, until a cathartic was administered on the fourth or fifth day. This gave rise to a very abundant fecal discharge, which continued for several days. It gradually diminished after this period, and the sinus finally closed in about six weeks from the time of operation. He has since been in perfect health.

Three years later the following case was observed, which clearly demonstrated the relationship between an acute perforative diverticulitis of the sigmoid and a generalized fatal peritonitis:

A. W., aged fifty-four years, was admitted to the Roosevelt Hospital in August, 1905, suffering with the evidences of a rapidly developing general peritonitis. Four days before admission the patient had experienced severe pain in the abdomen, which was soon followed by nausea and vomiting. The pain at first was located in the region of the umbilicus, but later shifted to the lower abdomen, and finally became localized. In the beginning of the attack there was little or no fever, the chief complaint being the irritability of the stomach, which led to the belief on the part of his attendants that the patient was simply suffering from an acute attack of indigestion. Accordingly, a cathartic was administered, and other appropriate treatment advised. On the third day the symptoms became worse: there was more pain in the lower abdomen, a gradual rise in temperature, and a progressive increase in the pulse rate. The abdominal tenderness increased and a gradual distention appeared, with a renewal of the vomiting and a rapidly advancing prostration.

When admitted to the hospital his temperature was 103°; pulse, 128; leukocytes, 16,000. The abdomen was greatly distended, everywhere tender, and tympanitic to percussion. Muscular rigidity was well marked, as much on the left as on the right side. Examination of the heart and lungs was negative. The urine was loaded with albumin and granular and hyaline casts.

Acting on the belief that we had to do with a perforating appendicitis with spreading peritonitis, under ether anesthesia, an incision was made through the right rectus muscle. As soon as the peritoneum was opened there escaped a quantity of thin, foul-smelling pus. The intestines were deeply injected and matted together, and in places covered with a fibrinous exudate. The appendix was sought for and found to be intact and no more inflamed than the surrounding viscera. As the pus seemed to well up from the pelvis, the incision was enlarged, and the intestines drawn upward out of the cavity. At the bottom of this space we found an oval fecal concretion somewhat larger than a date seed; and, nearby, a deeply congested loop of the sigmoid, on the free border of which was the gangrenous remains of a small perforated diverticulum. The perforation was closed by Lembert sutures, the entire cavity washed out with normal salt solution, the wound partly united, and a cigarette drain carried down to the bottom of the pelvis.

The patient rallied well from the operation, but after forty-eight hours of improvement the vomiting returned, the abdomenn became more distended, the urine diminished in quantity, the temperature and pulse rose, and he became progressively weaker. All attempts to move the bowels were unavailing, and he died at the end of three days.

The last two cases prove conclusively the relationship between acute perforative diverticulitis of the sigmoid and a localized intra-abdominal abscess in one instance, and a spreading peritonitis in the other. They suggest also the probability that the concretion in each instance acted as a predisposing cause of the perforation.

That the condition is rare there can be no question, but that it is far more frequent than one would suppose from the scanty reference to it in medical and surgical literature I believe to be highly probable. While in four of my cases the relationship between a diverticulitis and the abscess is not proved, still the similarity in symptoms between them and the last two cases is so striking as to lead to the belief that they might well have had a similar origin, especially as it is difficult to account for their origin by any other theory.

THE ESTIMATION OF THE FUNCTIONAL POWER OF THE CARDIOVASCULAR APPARATUS.

A PRELIMINARY REPORT.

By Richard C. Cabot, M.D.,
and
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of Boston, Massachusetts.

Probably we are all fairly well satisfied with the ordinary methods for examining the heart and vessels in all cases in which, as we say, we *find something;* for example, in obvious valvular leaks, obstructions, and vascular degenerations. But there is a large group of cases in which we find nothing by auscultation and percussion, nor by the study of the palpable vessels and the examination of the peripheral parts and internal organs for evidence of passive congestion, even when these methods are supplemented by radioscopic examinations and blood-pressure measurements. "Heart negative" we often write; but we feel by no means sure that the heart is as sound as our examinations are negative.

Especially if we are interested, as no doubt we are, more in the question—"What can the heart do"—than in the question—"What anatomical lesions exist in it"—we need some supplement to the methods of examination above listed. In the differential diagnosis between cardiac neuroses and permanent organic damages of one or another description, between the nervous heart and the weakened heart, we are often in a quandary. Or, again, when we are not seriously in doubt about the diagnosis, but desire to know *how far* a patient's convalescence has advanced with respect to cardiac power, or *how far* a known lesion has weakened the cardiovascular apparatus, we sometimes find the ordinary methods of examination at fault. In such cases, following Rosenbach, we supplement them more or less by experiments. We say: "Work a bit, and see how much it tires you," or "Do you get out of breath when you go upstairs?" "How many pillows do you have to have at night?" "Do you have palpitation when you hurry?" These are very rough-and-ready methods, yet they sometimes yield us information of more value than our direct physical examination. In the attempt to render more accurate the data at which we aim by questions such as those just suggested, a number of procedures have recently been suggested by German writers. It is the purpose of this paper to describe some of these methods, and to report briefly our own experience with them.

I. The Effects of Changed Position. It has been noticed by Schapiro and others that the normal difference between the pulse rate in a standing position and the lower pulse rate in recumbent position disappears when the heart is seriously weakened. In 100 cases we have tested this simple procedure—by counting the pulse first while the patient stands, and then while he lies flat: we find that the normal slowing of seven to fifteen beats per minute, which recumbency produces in healthy persons, is diminished or altogether lost in cases of uncompensated valvular disease, and when the heart is seriously weakened by any cause. We have not yet studied this test extensively enough to know whether it would help us to recognize cases of *incipient* cardiac weakness not appreciable by the ordinary methods of examining the heart.

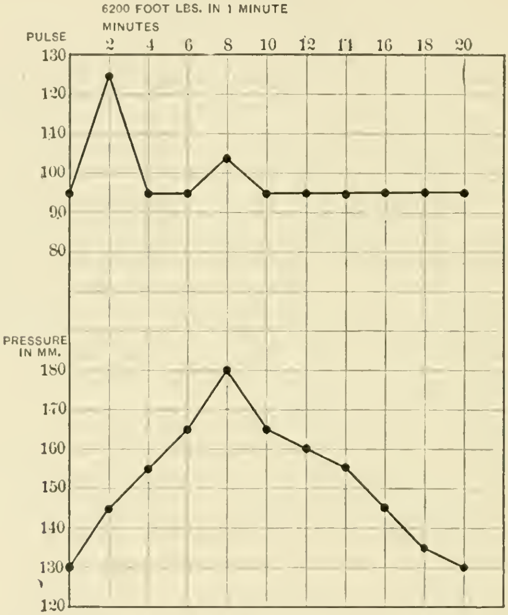

Chart I (which in our experiments represents the commonest type of normal reaction, or "*Erholung*" after exertion), shows a sudden rise in the pulse rate from 95 to 125 immediately following 6200 foot-pounds of work, accomplished in one minute by stair climbing. Four minutes after the performance of this work, when the pulse rate had fallen again to normal, the blood pressure had *risen* to 155, *or* 10 *mm. of mercury higher than it was when the pulse was most accelerated.* At the end of six minutes the pulse was still normal, but the blood pressure had risen to 165. At the end of eight minutes the blood pressure, continuing its rise, had reached 180 mm. After this a gradual fall in blood pressure occurred, the normal being reached only at the end of twenty minutes, although the pulse rate was normal in four minutes. Case 81,623. Normal. Aged twenty-four years.

II. Herz's Test.—Another procedure, suggested by Herz,[1] is called by him the Self-checking Test (*Selbsthemmungsprobe*). He counts the pulse over a period long enough to assure a reasonably constant rate per minute. He then has a patient sit down and very slowly flex and extend the right forearm, putting all the while his full attention upon the movement, but endeavoring to avoid any contraction of the muscles. During this test the physician keeps his left hand supporting the patient's elbow, while his right hand, grasping the patient's wrist, directs the slow movements of flexion and extension, without, however, assisting or hindering them in any way.

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[1] Deut. med. Woch., 1905, xxxi, 215.

Herz asserts that while in normal hearts this procedure exerts no influence upon the rate of the heart, a weakened heart is notably slowed. We have tested this method on 125 cases of miscellaneous affections, such as are seen in our out-patient clinic, and in a general way we have been able to confirm Herz's results. We have found that the great majority of cases in which the cardiovascular system shows no abnormality when examined by the ordinary methods, are also negative to Herz's test. A certain proportion of hearts which by other methods show evidence of weakness, react as Herz describes to the self-checking test; that is, the pulse slows down from 5 to 20 beats. In carrying out the test,

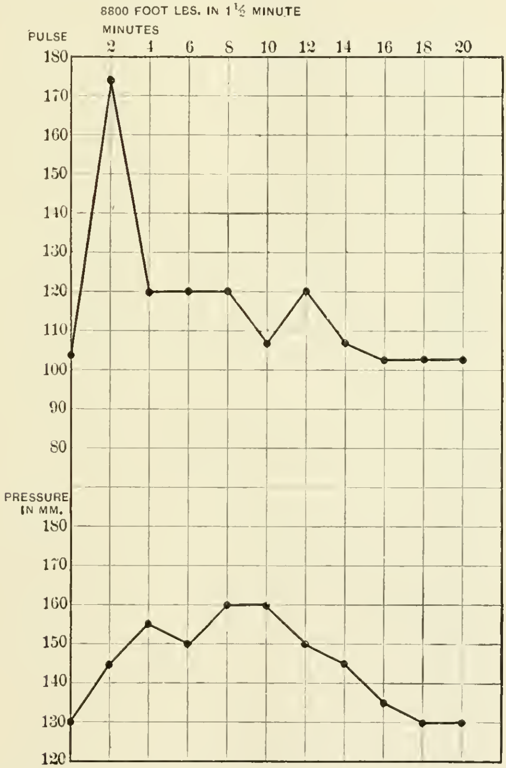

Chart II shows but slight variations from Chart I. In both of these cases the heart was young and apparently strong. Case 84,901. Normal. Aged nineteen years.

we have made it a rule that extension shall consume a full minute's time, and flexion also a minute. We are encouraged to follow this test further, although we have not as yet obtained any striking results with it.

III. Levy's Test. Katzenstein's test[2] is based upon the well-known fact that if we increase the peripheral resistance opposed to

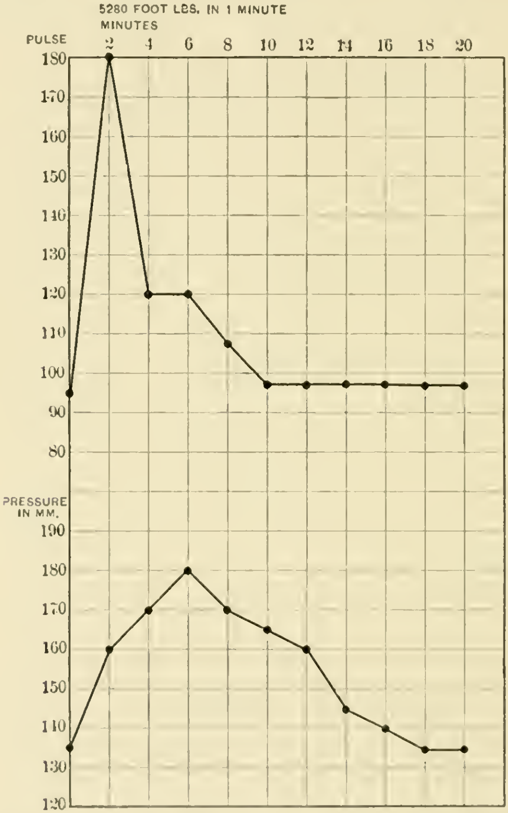

Chart III from a case of mitral stenosis, with perfect compensation, shows essentially normal conditions so far as this test is concerned. The rise in pulse rate, after 5280 foot-pounds of work performed in one minute, was more marked than in the normal heart, but the two charts are otherwise practically alike. Case 5354. Mitral stenosis. Aged twenty-one years.

the heart's action by compressing the abdominal aorta (in laboratory animals), a marked rise in blood pressure occurs. Anyone who is familiar with experiments on animals must have often seen the temporary resuscitation of a failing heart by compression of the abdominal aorta, and must also have noted that if the heart is quite exhausted, whether by hemorrhage or for other reasons, compression of the aorta is without its usual effect. In human beings

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[2] Berl. klin. Woch., 1905, Nrs. 15 und 16.

Katzenstein imitates this familiar laboratory procedure by digitally compressing both femoral arteries at Poupart's ligament, and then noting the effects upon systolic blood pressure in the brachial artery. In normal persons this procedure is followed within two or three minutes by a rise of from 5 to 15 mm. of mercury in the systolic blood pressure. When the heart is hypertrophied (Levy[3] says) the rise is even more marked, while if the heart is weakened by any cause there may be little or no rise of blood pressure. Levy claims that no pain is produced by this procedure, but admits that it is useless in neurotic persons, since in them any such manipulation will suffice to raise blood pressure. We have made but few tests

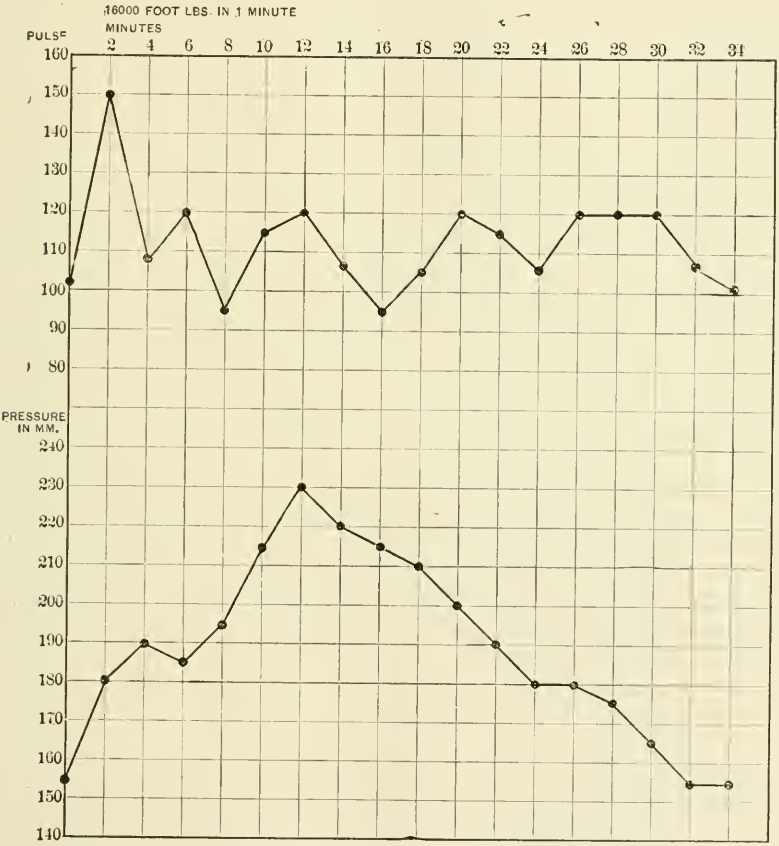

Chart IV shows the pulse and blood pressure curves from a case of weakened heart in a woman of forty-two. It will be noted that the "Erholung" is here *retarded*, so that the highest blood pressure is not reached until the end of ten minutes, while the return to normal occupies thirty-four minutes in all. Case 82,475. Myocarditis. Aged forty-two years.

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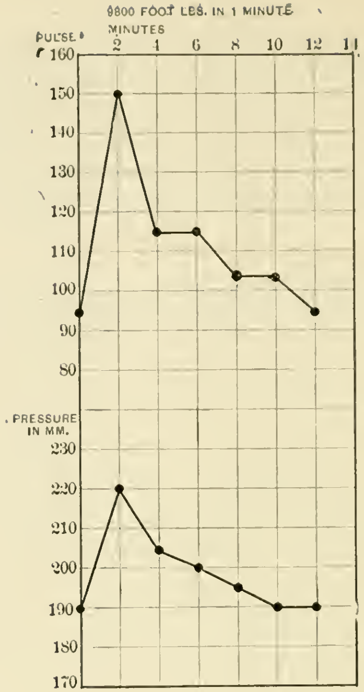
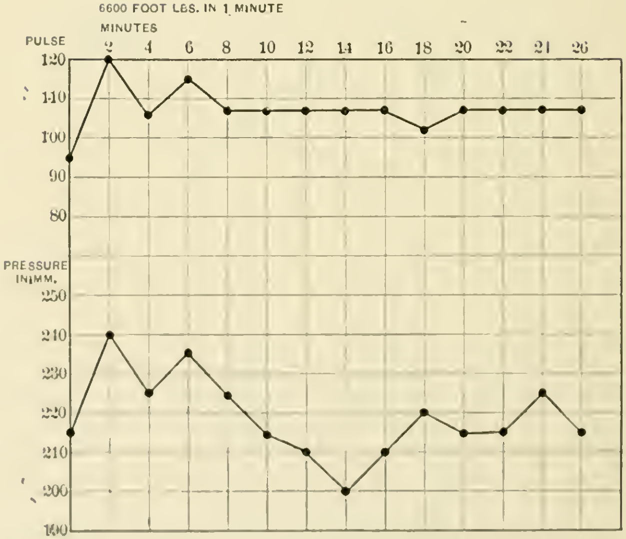
[3] Zeit. f. klin. Med., 1906, lx, 1 und 2.





CHARTS V and VI illustrate the curves which we have found in a number of more seriously weakened hearts in older persons, with higher blood pressure than any of those shown in the previous charts. In these two patients a slight rise in blood pressure was simultaneous with the rise in pulse; thence it gradually declined to normal in Chart IV, and slightly below normal in Chart V. The characteristic increase of blood pressure after the pulse rate has returned to the normal level is absent in these two charts. Chart V. Case 84,478. Arteriosclerosis. Aged fifty years. Chart VI. Case 84,516. Hypertension. Nephritis? Aged sixty years of this procedure, as we have been unable to perform it without causing considerable pain, the effect of which would naturally be to raise blood pressure independent of any other cause.

IV. GRÄUPNER'S TEST. The test to which we have given the most attention is one of those elaborated by Gräupner[4] in several recent papers. Gräupner, who is a "Bad-Arzt" at Nauheim, found in the course of exercises prescribed as part of his treatment of cardiac cases that the reaction of weakened hearts after a measured amount of work differed distinctly from that of normal hearts. It is, of course, a familiar fact that the pulse rate and the systolic blood pressure rise after exertion, and that if exertion is sufficiently prolonged and arduous, both these figures may fall to or below the normal; but the phenomenon to which Gräupner has called special attention is quite different from this. Gräupner found that *after the pulse rate has risen and again fallen* to normal, as result of exertion, *the systolic blood pressure begins to rise,* reaches its maximum some minutes later than the pulse rate, and gradually falls thereafter to, or sometimes below, the normal. The phenomenon just described is that found by Gräupner in normal cases, and designated as the "*normal Erholung.*" In weakened hearts, if the damage is but slight, the "*Erholung*" still occurs, but is delayed and perhaps diminished in degree. In seriously weakened hearts there may be no "Erholung" whatever, the blood pressure declining from the start, then gradually re-ascending to the normal.

The main outlines of Gräupner's contention may be easily verified by anyone. Run quickly up two flights of stairs, and then stop and count your pulse. After the immediate acceleration is past or during the slowing of the pulse following it, you will note that the heart beat and the strength of the pulse become markedly exaggerated. One feels the "thump-thump" of the heart against the ribs much more strongly after the pulse has almost or quite reached its normal rate than during the period when the pulse is most accelerated.

In performing this test, Gräupner used a weight-and-pulley ergometer, but in our clinic this was not convenient, hence we substituted a measured amount of stair-climbing. We reckoned the number of foot-pounds of work by the height ascended, computed in feet and multiplied by the number of pounds which the individual weighed. The amount of work in foot-pounds has, therefore, varied considerably, according to the weight of the individual, although the number of stairs climbed has usually been the same. As regards the phenomenon designated by Gräupner as the "*normal Erholung,*" we can verify his findings, and we likewise agree with him in the results of our experiments upon seriously weakened hearts. In some of the cases believed by us from ordinary examina-

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[4] Collected in his monograph: Die Messung der Herzkraft, München, 1905, Otto Gmelin.

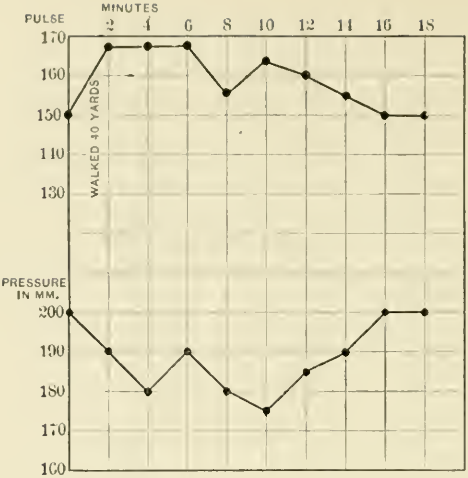

Chart VII shows the results obtained in a case of ruptured compensation, the patient being too ill to climb any stairs, markedly dropsical and cyanotic. Blood pressure *fell from the start* (simultaneously with a rise in pulse) and took eighteen minutes of perfect rest to bring it back to the (very high) point indicated at the beginning of the experiment. Myocardial weakness, cyanosis, etc. Aged forty-eight years.

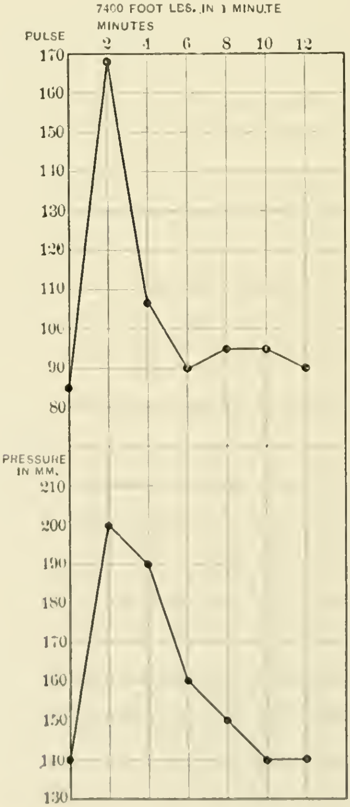

Chart VIII.—Case 84,318. Phthisis; "heart negative." Aged forty-five years.

tion of the heart to be normal, there was considerable variation from the ordinary curve of blood pressure after exertion. On the other hand, various cases of valvular disease with good compensation showed, as might have been expected, a normal curve. (See Chart II.)

The important question regarding this and the other tests above described is, of course, "Do they help us to recognize the presence or the degree of cardiac weakening not appreciable by the methods already in general use?" We do not feel prepared to decide this question from the 75 experiments which we have been able to make in the four months during which we have been engaged in this work; but we have felt distinctly encouraged to continue the work, and in actual practice we find ourselves instinctively turning to Gräupner's test to verify our doubtful diagnoses of weakened heart, or to support us in the belief that the heart is normal when the patient has complained of subjective disturbances which might have been interpreted as evidence of passing congestion.

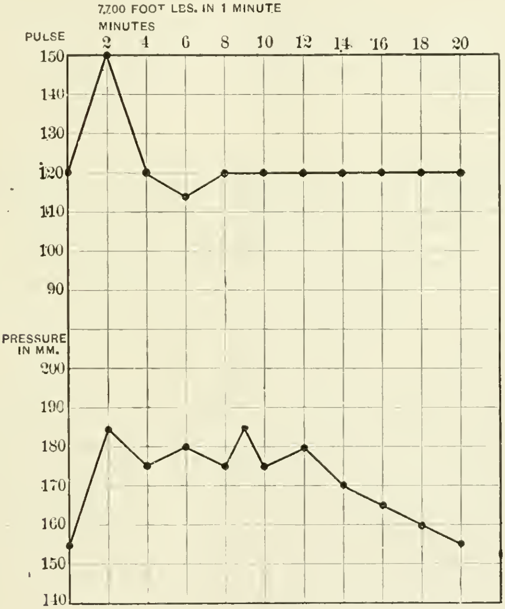

CHART IX.—Case 84,131. Palpitation. Nervousness. Myocarditis. Aged fifty-two years.

We suppose that the phenomenon just described depends primarily upon the amount of elasticity and contractile power in the arterial system; and, secondly, upon the ability of the heart to react upon the stimulus of increased peripheral resistance by beating more strongly. In all our measurements the effect of age in diminishing arterial elasticity is strongly suggested; but we have not yet seen reason to believe that this is the only factor concerned.

SUMMARY AND CONCLUSIONS.

1. For diagnosis, prognosis, and treatment it is important to supplement the current methods of cardiac examination by others planned to measure the functional capacity of the cardiovascular apparatus.
2. Several such methods have recently been suggested by Germans.
3. Our experience with these methods, and especially with that of Gräupner, leads us to hope that ground has been gained in the direction of functional cardiac diagnosis.

THE TREATMENT OF ILLUMINATING GAS POISONING BY THE DIRECT TRANSFUSION OF BLOOD.

By George W. Crile, M.D.,
and
Carl Lenhart, M.D.,
of Cleveland, Ohio.

Occurrence. At least the main toxic factor in artificial illuminating (coal) gas poisoning is carbon monoxide. Carbon monoxide constitutes 6 to 10 per cent. of illuminating gas. It occurs in the so-called "water gas" to the extent of 30 to 40 per cent. It is the important toxic constituent of the "after damp" of mines, and of the gas from charcoal and coal stoves and from blast furnaces.

Mode of Action. Its action is purely that of asphyxia. It combines with hemoglobin, displacing the oxygen. Its affinity for hemoglobin is 300 times that of oxygen. The resulting compound is not perfectly stable, so that when oxygen is present in very great excess, carbonic oxidhemoglobin will be decomposed and oxyhemoglobin formed. Hence the rational treatment of patients by oxygen inhalation.

Haldane placed animals (mice) in oxygen under a pressure of two atmospheres, so that sufficient oxygen went into simple solution in the blood serum to support life independently of the hemoglobin. Sufficient carbon monoxide was present completely to saturate their hemoglobin. Under these conditions the mice remained normal as to symptoms, showing that the carbon monoxide has no direct toxic action. When the pressure was removed and the mice put out in the air they died with symptoms of asphyxia.

Haldane claims that in recovery carbon monoxide is given off from the lungs, as such, and is not destroyed or oxidized in the body.

Symptoms. To summarize: there is stimulation of the central nervous system, followed by paralysis, the order being—brain, spinal cord, medulla. To detail:

1. There is cerebral excitement.
2. There is stimulation of the medulla: (*a*) Dilatation of the cutaneous vessels with flushing. The lips are pink. If the poisoning be very acute, there may be cyanosis, due to the fact that the venous blood does not yet contain much carbon monoxide. (*b*) The blood pressure rises, due to the stimulation of the vasomotor centre. There may be ecchymoses. (*c*) The heart is first slowed, due to vagus stimulation. (*d*) Respiration is dyspnœic. (*e*) There may be vertigo, vomiting, and mydriasis.
3. Paralysis of the medullary centres follows: (*a*) The blood pressure falls, due to vasomotor paralysis. (*b*) The heart rate is increased, due to vagus paralysis. (*c*) Paralysis of respiration (cause of death).
4. Following cerebral excitement there is unconsciousness, anesthesia, and coma.
5. There are often convulsions—from below up.
6. At death the hemoglobin is usually combined with the carbon monoxide to the extent of 60 to 80 per cent.
7. In dilute solutions the blood is pink and presents a characteristic spectrum.
8. Late symptoms and after-effect: these are quite lasting in man. They may be due in part to impurities in the gas. These late symptoms are prolonged unconsciousness or mental dulness, headache, nausea, spastic condition of the limbs, and epileptiform seizures. There may be loss of memory and mental derangement for months. Pneumonia may follow.

If the patients are breathing well when rescued, the blood is probably freed from carbon monoxide in about three hours. The danger from the condition of the blood itself is probably over in an hour, that is, if there is good respiration. But when the respiration is shallow and meager, this time is, of course, much extended. The late symptoms must be due, not to the blood condition directly (for this may be normal in a few hours), but to serious and more or less lasting injury to the nervous and other tissues. Hence, any measure tending to shorten the period during which the blood is impaired will tend to prevent the late symptoms due to tissue injury.

The above statements have been largely based upon Dr. Sollman's *Pharmacology* and Haldane's article in Hale White's *Pharmacology*.

Through the kindness of Drs. A. B. Kanavel, and H. M. Richter we have received by private communication the report of 16 cases of gas poisoning, occurring in the various services of Cook County Hospital, Chicago. Statistics based on these cases may render the matter more concrete and emphasize the need of treatment beyond that hitherto practised.

Number of cases 16.

Color. Mentioned 14 times. Cherry-red, 2; flushed, 1; cyanotic, 5; pale, 1; negative, 5.

Breathing. Dyspnœa, 15; apnœa, 1 (this case was associated with morphine poisoning).

Coma, 15; mentally clear, 1.

Pupils react to light, 7 out of 11 times tested; 4 did not.

Musculature. 3 had general tetaniform convulsions; 1, muscular tremor; 2, general rigidity; 2, stiff jaws.

Pulse, ran from 96 to 200. 11 cases had as their highest pulse rate 120 to 160.

Reflexes (knee-jerks). Mentioned 12 times. Present 9 times, exaggerated once, absent twice.

Temperature. Classified according to highest reached: 98° to 99°, 1; 101° to 102°, 1; 102° to 103°, 3; 103° to 104°, 1; 104° to 105°, 1; 105° to 106°, 2; 106° to 107°, 3; 107° to 108°, 2; 109.2°, 1 case.

TREATMENT. All received the usual stimulant drugs; 10 received oxygen inhalations; 4 were bled 10, 6, 4, and 8 ounces; 5 received intravenous saline injections; and all 16 cases died.

(*a*) Artificial respiration. It is needless to give reasons for its use.

(*b*) Fresh air.

(*c*) Oxygen is certainly indicated, not to oxidize the carbon monoxide, but by being in great excess gradually to displace it.

(*d*) Heat. Many persons have collapsed on being taken into the cold air, hence, it would seem better to institute treatment in a warm room.

(*e*) Patients have often died from exertion; for example, from violent endeavor to escape from a mine. There is probably acute dilatation of the heart in these cases.

(*f*) Stimulants such as camphor, caffeine, digitalis, strychnine, etc.

(*g*) Venesection. This method is rational when combined with the injection of salt solution or blood. As to its use alone, it is condemned by most of the older writers on the ground that it further depletes a patient already in a low condition. On the other hand, Halstead reported 2 cases in which he thought it did good. Reviewing the literature, Halstead thought that no case had been saved by transfusion when venesection had failed. This was in 1884.

(*h*) Transfusion of defibrinated blood by the older methods, together with venesection. This is mainly of historical interest. The theory was that venesection rid the body of some of the poison and the transfusion would replace the lost hemoglobin with fresh hemoglobin. The basis of this procedure was the experimental work of Kühne in the 60's of the last century. In a monograph by Willy Sachs,[1] his work is summarized as follows:

[1] Die Kohlenoxyde Vergiftung, 1900.

1. Animals poisoned until eye reflex is absent recover spontaneously, provided respiration is not reduced below 2 per minute.
2. If respiration be reduced to 1 a minute, they do not recover unaided. If bled smartly they usually recover.
3. If respiration stop for a few minutes, they do not recover even though the heart is beating well, and even if they be bled and given artificial respiration.
4. Even after respiration has stopped several minutes (1 case seven minutes) when there is no peripheral pulse, no heart beat detected through the chest wall, and often with the animal in convulsions, transfusion of blood will bring back life.

There is this criticism to be offered concerning Kühne's experiments. He groups his cases largely on a basis of the rate of respiration. We have found that this is very irregular, so much so that one could never definitely say that the respiration was 2 or 1 a minute. In fact it is difficult to say when respiration has finally stopped. A large majority of the dogs stopped breathing for two or even five minutes and then began again with a fairly good quality of respiration. So we think that many of Kühne's good results were due to the fact that he began his measures during this first interval of cessation of respiration; and his dogs were not nearly so dead as he thought.

On the basis of these experiments, clinical cases of gas poisoning were transfused. This was in the 70's and 80's. Favorable cases were reported by Utuhart, Badt, Bouveret, Lühe, Rüdinger, Kahler, and Leyden. To mention a few instances, Von Heuter (1870) had a patient who was unconscious and upon whom artificial respiration had to be maintained. 500 c.c. of defibrinated blood were transfused. The patient regained consciousness in a few hours and recovered.

Leyden had a patient found in coma at 5 A.M. Venesection and artificial respiration brought no improvement. At 11.15 A.M. he had no corneal reflex; respiration was slow, irregular, and shallow; pulse bad. At 1 P.M. 250 c.c. of defibrinated human blood was transfused. At 6 P.M. he turned over in bed. At 4 A.M. he wakened, drank milk, and went on to complete recovery.

Friedberg, while he reported 3 unfavorable cases, still favored transfusion. He thought its failure due either to insufficient and poor blood, or to great injury to the nutrition of the respiratory and circulatory systems.

Albert mentions 2 cases. One had re-acted to none of the usual stimulants; heart was barely perceptible. Was transfused, recovered and could stand the next day. Another case of seemingly the same degree of intoxication was not transfused. This patient was three days unconscious. He finally lived but had the late symptoms, nervous trouble, for a month. Albert, therefore, favored transfusion.

Opposed to these favorable views were such men as Hunter and von Bergman, who thought that the danger of thrombosis and embolism outweighed any advantage that transfusion might have. Among pharmacologists, Kobert favored and Kunkle opposed transfusion on the same grounds.

(*i*) Refusion of blood. Halstead, in 1884, recorded a case treated by this method. Patient was comatose, respiration 28, shallow, pulse, 96, small, easily compressible. There was general rigidity. 512 c.c. of blood was withdrawn from the right radial artery, defibrinated and strained. 288 c.c. was reinfused centripetally into the artery. The patient made a good recovery.

(*j*) Saline intravenous infusion. In the 80's the transfusion of blood was forsaken and everyone turned to the new physiological saline solution. We have found the following recorded:

I. Jersey[2] reported a patient in collapse for twenty-four hours. Was bled 8 ounces and given 11 ounces of salt solution intravenously. Patient recovered.

II. Wilkie[3] reported 2 cases.

1. Bled 13 ounces; solt solution 13 ounces; recovery.

2. Bled 6 ounces; salt solution 13 ounces; death. Marked temporary improvement.

III. Schruber reports a patient unconscious and in convulsions on the second day. Eleven and one-half hours after having been given saline infusion he began to speak and finally recovered.

IV. Von Gordon reports 3 cases:

1. Patient unconscious, slow stertorous breathing, pulse weak and irregular, 72, reflexes lost. Bled 300 c.c.; saline 400 c.c.; recovered.

2. Patient unconscious, respiration irregular, pulse dicrotic, irregular, 120. Convulsions. Bled 300 c.c.; salt solution 300 c.c.; recovered.

3. Patient unconscious, convulsions, dyspnœa. Bled 165 c.c.; salt solution 300 c.c.; recovered.

V, VI, VII, and VIII. From Cook County Hospital cases, all terminated fatally.

Our review shows:

(*a*) The older experimental work seems faulty and insufficient.

(*b*) The clinical evidence is variable but, on the whole, leads one to have hope that the newer method of transfusion of blood will be of considerable value in these cases.

Our own research was instituted to test the value of the various therapeutic measures suggested, especially blood transfusion. Dogs were used. Ether was the anesthetic. Various forms of apparatus were used. The last one used was a large, air-tight, iron tube, into which gas and air entered through water bottles, by the bubbling of which one could make a rough guess as to the amount entering. No attempt was made to determine the exact proportion of gas in

[2] Cited by Bell New York Med. Record, 1884.

[3] Ibid.

the mixture. It was decided, owing to the varying susceptibility of the animals, that the best basis for comparison of therapeutic measures would not be the percentage of gas, its quantity, or its time of action (that is within narrow limits), but rather the exact condition of the animal as determined according to ordinary clinical means, and especially by a manometric tracing of the blood pressure. Consequently, the carotid artery was attached to a manometer with a style writing on a revolving drum, in the usual way. A tracheal cannula was inserted and connected to a T-tube. The two branches of the T-tube were connected to valves made of fingers of rubber gloves, set in small flasks. One valve opened with inspiration, the other with expiration. The valves were connected respectively with the gas tank and the outside air, so that the dog inspired the gas mixture and expired into the outside air. The dogs were thus poisoned, and when the blood pressure reached a certain low point the various therapeutic measures were instituted and their results compared.

It would require too much time to give the history of individual experiments. A few statistics of the results may be of interest.

In all cases respiration had stopped. In the 16 cases recorded the heart had stopped in 8 cases before any measures of resuscitation were taken. Immediately the heart stopped, however, these measures had to be begun. Resuscitation was never accomplished after the heart stopped for any length of time. In 6 cases the heart had almost stopped, with a blood pressure varying from 8 to 20, with an average a little under 14 mm.—certainly a low blood pressure. In 2 cases the blood pressure was 54 and 44 mm. One was saved by transfusion, the other died notwithstanding the giving of saline.

(*a*) Animals treated by bleeding, manipulation of the heart, and artificial respiration. One experiment. Died.

(*b*) Animals treated by bleeding, intravenous saline, manipulation of the heart, and artificial respiration. Three experiments; 3 died.

(*c*) Animals simply treated by manipulation of heart and artificial respiration. Two experiments; 2 died.

(*d*) Animals treated by blood transfusion, bleeding, manipulation of the heart, and artificial respiration. Ten experiments; 7 lived, 3 died.

Reduced to the low degree of blood pressure, 7 out of 10 were rescued by transfusion of blood; 6 other dogs treated by the various other methods all died.

Of cases in which the heart stopped:

1. Blood transfusion saved 3 out of 6.
2. Intravenous saline. None saved; 1 died.
3. Simple manipulation. None saved; 2 died.

Of cases in which the heart almost stopped:

1. Blood transfusion saved 4; 1 died.
2. Intravenous saline. None saved; 2 died.

These experiments include only those done while taking a blood-pressure record on the manometer, so that the exact condition of the dog could be constantly watched.

A large number of experiments have been discarded, in which no drum record was taken, because it was found that if the exact status of the dog was judged clinically, that is, by pulse rate, stethoscope, etc., the results were so variable and so far from following any rule, so different from the above results, that one was forced to the conclusion that the starting point for initiating the various therapeutic measures was not being made reasonably the same. Hence, the conclusions could not be exact enough to be of value. Instead of an experiment with exact limitations, we were simply reproducing clinical cases, as seen in the hospital.

By recoveries we mean cases in which the blood pressure comes back approximately to the point where it started and remains fairly constant for an hour or so, and in which the respiration becomes spontaneous and of good quality again and the reflexes reappear. Actual recoveries were not done because of the difficulty of maintaining asepsis with our present means, especially while taking blood pressure records with a cannula in the carotid artery.

CONCLUSIONS. 1. Blood transfusion seems to be of greater therapeutic worth than the other measures considered.

2. For the animal to recover, it is necessary to begin transfusing immediately the heart stops beating. It is not efficacious after the heart stops finally, as, for example, is adrenalin in chloroform death.

THE EXAMINATION OF THE FECES FOR OCCULT BLOOD, WITH SPECIAL REFERENCE TO THE VALUE OF THE BENZIDIN TEST.

By Edward H. Goodman, M.D.,
OF PHILADELPHIA.

(From the private laboratory of Dr. John H. Musser, Philadelphia.)

Since Boas,[1] in 1903, directed the attention of the medical world to the significance of small hemorrhages (unrecognizable macroscopically and called by him "occult") from the gastro-intestinal tract, the study of the feces from this viewpoint has become one of the most important of the routine clinical laboratory examinations. Since time immemorial, until comparatively recent years, the finding of blood arising from the gastro-intestinal tract was a very crude procedure and limited to but one means, the sense of vision. Either the hemorrhage must have been high up and have shown itself from the mouth as bright red blood, or as so-called "coffee-ground vomit;" it must have been low down (hemorrhoids, ulcers, etc.), when it would make its appearance in the feces as easily recognizable streaking; or the hemorrhage must have been intestinal, and comparatively large, in order to lend the feces the classical "tarry" appearance. If this macroscopic examination sought substantiation in the microscope, we fear the observers were often doomed to grievous disappointment, for inasmuch as most of the red blood cells are rapidly destroyed in the upper portion of the intestinal tract, their appearance microscopically in the feces must have been one of great infrequency and rarity.

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[1] Deut. med. Woch., 1903, No. 17.

We now know that only exceptionally has the color of the feces any relation to the presence or absence of blood. We have all seen the typical fatty stool, of very light color and offensive odor, yielding a beautiful chemical test for blood, whereas, with less accurate means at their disposal the older observers would have been disposed to regard the stool negatively as regards blood. It is this hidden, insidious bleeding which, since Boas' article has received such attention, that we must now regard and not direct all our thoughts toward the macroscopic bleeding. While the latter is, of course, important, the former is much more so. It is well known that a large profuse hemorrhage is often preceded by a very small, macroscopically not recognizable, bleeding, hence, if this can be detected, and properly treated, a large hemorrhage may often be averted. On the other hand, when a routine examination of the feces is not made regularly for each patient, the macroscopic appearance of the stool may often lead to a more minute examination. If, for example, the case is one of colica mucosa, the macroscopic examination discloses ribbon-like pieces and meshes of mucus, which, if examined microscopically, are seen to be covered with red blood cells. In this case, and in some cases of intestinal carcinoma, the microscopic examination is seen to be more important than a routine blood test, for it leads to a diagnosis, whereas, the occult blood test at the best is but an aid in determining the nature of the morbid process. So, because we have a reasonably simple and exact test, we must remember not to put all our reliance on it alone, but must call to our assistance simpler methods, which, in this era of scientific research, with its accompanying accurate methods of examination, reliable instruments of precision, and unimpeachable chemical tests, we are too likely to overlook.

The methods employed today for the detection of macroscopically invisible blood are based on a test of a single chemical constituent of blood, namely, blood coloring matter, or one of its near substitution products. It is unnecessary to explain each test in detail, as descriptions may be found in the various text-books, and in a very careful and instructive monograph by Schumm,[2] from whom I have derived much benefit in preparing this paper. It will suffice to mention only the tests now used:

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[2] Die Untersuchung der Feces auf Blut, Jena, 1906.

The *Teichmann Hämin test* (rarely employed).

The *Nencki-Koberts acetonhämin test*, in which the diagnosis is made by a microscopic examination of the hämin crystals. The feces are first freed from fat by means of alcohol followed by ether, and after adding a drop of concentrated hydrochloric acid, the stool is ground up with aceton. The feces are now boiled and filtered hot into a watch crystal, a few drops only being collected on a slide. On rapid evaporation, the hämin crystals are seen on the latter as "extremely characteristic, long, curved, hair-like crystals" (Nencki), while on slower evaporation in the watch glass they are thicker. Schumm has tried this test, but does not advise it for clinical purposes.

The Spectroscopic Examination. For this purpose the acetic acid-ether extract obtained while doing Weber's test may be used, or the suggestion of Schmilinsky[3] may be followed. Schmilinsky recommends the conversion of hematin into hematoporphyrin by grinding up a small portion of feces with water and adding from a few drops to a few cubic centimeters of concentrated sulphuric acid. By using a vessel with flat sides, the characteristic absorption lines may be seen. He recommends in case of negative findings the use of the guaiac test, as being more sensitive. If the characteristic lines are present there can be no doubt about the presence of blood, as, to our knowledge, no substance but blood gives a similar spectrum. I have had very little experience with the use of the spectroscope in looking for occult blood and can give no advice based on personal observation.

The tests that are generally employed at the present time are the guaiac (Weber's) and the aloin (Rossel's) tests.

Weber's Test. Weber's own directions are as follows: A large portion of feces is ground up with water, to which one-third its volume of glacial acetic acid has been added, and this mixture is extracted with ether by shaking. After standing a short time, a few cubic centimeters of the acid-ether extract is poured off and ten drops of the tincture of guaiac and thirty drops of turpentine are added. In the presence of blood the mixture becomes blue violet, whereas a reddish-brown color with a shade in green is noted if blood be absent. The reaction becomes more pronounced if the blue color is extracted with chloroform, after first adding a little water to the acetic-acid-ether extract.

Weber speaks of some sources of error which are mentioned by Schumm, and the latter observer has found that frequently in following Weber's method an emulsion has formed which was often hard to remove, but he succeeded eventually with alcohol. He found it necessary sometimes to add alcohol, then ether, and then more alcohol. Boas has made the objection that alcohol sometimes produces the blue color, but Schumm, in thousands of examinations, says he has never seen alcohol alone give this color.

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[3] Munch. med. Woch., 1903, 2115.

Schmilinsky says that ozonized turpentine gives the characteristic color if it has stood in direct sunlight, which observation Schumm substantiates, but says the turpentine can be again brought to normal condition if put in a shady place. He recommends putting the turpentine in large open vessels, and letting it remain in a semi-dark cellar for weeks.

Rossel[4] was the first to recommend the removal of fat by drying the stool and then extracting with ether in a Soxhlet apparatus; Schumm very properly takes exception to this, as being a too complicated procedure for ordinary clinical purposes. v. Koziczkowsky[5] modifies Rossel's suggestion and shakes the stool with ether, or grinds it up with ether, pours off the ether, mixes the feces with glacial acetic acid, extracts with ether by shaking, and tests the etheral extract. This method also calls forth an objection from Schumm, who offers a modification of Weber's test for which he claims many advantages.

Schumm's Modification of Weber's Test. If the stool is thick, he takes 4 grams of feces (about the size of a small walnut) and grinds it up with equal parts of alcohol and ether, 30 c.c., in a glazed porcelain mortar. He filters through a pure open filter, covers the residue with alcohol and ether, and mixes by gently stirring with a glass rod. This is allowed to filter, covered with ether, and gently stirred. After the ether has filtered through he again washes with ether and again filters. In this way soluble coloring matter, neutral fat, fatty acids, and water are removed. The washing is complete when the filtrate is only slightly colored, and, if desired, the filtrate can be used for hemin crystals; otherwise, it is disregarded. The residue remaining on the filter, which, besides food-rests, contains soaps and coloring matter not soluble in neutral alcohol-ether, is covered (on the filter) with 4 c.c. of glacial acetic acid and mixed by gently and thoroughly stirring. After the greater portion has filtered, 4 c.c. of glacial acetic acid is poured on and stirred with the fecal residue. After most of the acid has been filtered, it is poured back on the filter and again mixed carefully. In order to avoid the annoying fumes of the acid, he advises covering the funnel with a watch glass.

By this method blood coloring matter is set free and the organic katalizers (oxydizing ferments) are destroyed. In the absence of blood coloring matter the filtrate is rather deeply colored. Several cubic centimeters of the glacial acetic acid filtrate is diluted with its volume of ether, half the volume of distilled water is added, and the whole taken. If the fluids do not separate add some alcohol, ether, or water.

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[4] Deut. Arch. f. klin. Med., 1903, lxxvi, 505.

[5] Deut. med. Woch., 1904, xxxiii, 1198.

The ethereal solution is separated and again shaken with a small amount of water. This ethereal solution is to be tested in absolutely clean test tubes with guaiac tincture and turpentine. (Schumm warns against using test tubes already used for sugar tests; if no others are available, however, they may be used if washed with acetic acid and alcohol.) In the presence of hematin the color is more or less blue, violet, reddish-violet, as well as greenish-blue. Schumm claims that the Weber test, even in the presence of not inconsiderable amounts of blood, sometimes gives only a rapidly disappearing violet coloring, whereas, in the above modification, the color is slower in appearing, but is also more lasting. For thin stools the method is the same, only larger amounts must be used. Schumm recognizes the fact that in his modification a certain amount of blood is lost, but claims the quantity is inconsiderable and may be disregarded; as a control of his modifications he recommends the use of the spectroscope, which gives, to a certain extent, the quantity of blood present.

The question of just what shade shall be regarded as indicating the presence of blood seems to have been the source of much confusion in the reports of various authors who have studied the Weber test. Schmilinsky says not only the *blue* color is positive, but also the various other related colors. He does not consider the green color absolutely indicative of blood, as the ether sometimes takes up this color from the feces, and this interferes with the test, but if the green color appears it should be regarded as suspicious, and the test should be made more exactly with less ether in order to get a concentrated hematin extract; the test should be repeated on the following day, and the spectroscope should be used as a control. Schloss[6] found, as did Hartmann, whom he quotes, that only the blue-violet color indicates blood, and that all other shades arise from some coloring matter in the feces, and in this view he is supported by Schumm. The latter gives the following colors as being positive for his modification: ½ minute, green; 1 minute, blue green; 2 minutes, violet.

The Aloin Test of Rossel. Rossel makes a solution of Barbadoes aloin by putting as much aloin as will go on a knife point in 3 to 5 c.c. of 60 to 70 per cent. alcohol. He extracts the fat from the feces and makes a glacial acetic acid-ether extract, adding to this 20 to 30 drops of old turpentine and 10 to 15 drops of the aloin solution. In the presence of blood there is a beautiful cherry-red color appearing in from five to ten minutes; whereas, in the absence of hematin, the color first shows itself in from one to two hours, and remains. He advises making a fresh aloin solution each time before using, and claims the test is as sensitive as the guaiac test, for, after making experiments on himself, marking off his feces with charcoal, he found a positive reaction with 10 c.c. of blood.

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[6] Arch. f. Verd.-krankh., 1904, x, 267.

Benzidin Test of O and R. Adler.[7] As given in the original paper of these authors the test was found to be inapplicable for clinical purposes, and two important modifications have been proposed.

The first is from Schumm, who cleanses the stool first with alcohol and ether, as in Weber's test, and extracts with glacial acetic acid. The extract is washed with 1 to 2 c.c. of water, the acid-ether extract is then mixed with 2 c.c. of benzidin solution and H_2O_2 added. The second modification, which I have used exclusively in my studies, is that practised by Schlesinger and Holst,[8] with but a few changes. The following is the method of Schlesinger and Holst:

1. A concentrated solution of benzidin is made by using as much benzidin as will go on the end of a knife in about 2 c.c. of glacial acetic acid. (For several tests, correspondingly more.) The benzidin is readily soluble, and the mixture should be shaken a few times.
2. A small piece of feces about the size of a pea is taken up on a glass rod and suspended by stirring in a test tube one-fifth full of water; the test tube is closed with cotton and the feces are boiled.
3. Ten to 12 drops of the benzidin solution are poured into a test tube and 2.5 to 3 c.c. H_2O_2 (3 per cent.) added.
4. To this are added 1 to 3 drops of the boiled feces, after mixing by slightly shaking.

In the presence of blood the color becomes green, blue green, or blue, the blue being more pronounced if more blood is present. Slight amounts show a reaction in one-quarter to one-half minute, and very small amounts always within two minutes. After five to fifteen minutes the color becomes a dirty dark red violet and remains so. Negative tests show no change even after standing twenty-four hours.

SENSITIVENESS OF THE GUAIAC, ALOIN, AND BENZIDIN TESTS. The guaiac and the aloin tests have been found to be about equal in sensitiveness and both respond to blood in the dilution of 1 to 25,000; whereas the benzidin test is positive in a dilution of 1 to 200,000 (Schumm and Westphal).

SOURCES OF ERROR IN THE VARIOUS TESTS. In making any test the source of error must be kept in mind and removed as far as possible before a test can be considered as having any importance.

Guaiac Test. Rossel says the main disturbing agent is iron, and the importance of discontinuing the use of iron while the feces are being examined for occult blood has been discussed by Adler, Schumm, and others.

Every worker on the subject of occult blood has come to the conclusion that meat in the ordinary diet very often gives the guaiac test. Schmilinsky, in 1903, recommended a diet of milk, with coffee or tea, milk soups, sugar, butter, bread, rice, sago, farina and oatmeal, eggs, etc. He examines the stools on the third day. v. Koziezkowsky uses a similar dietary, but advises against the use of much fat and vegetable (chlorophyll often disturbs), and he marks off the stool with charcoal in preference to carmine, which he thinks interferes with the test.

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[7] Ztschr. f. phys. Chem., 1904, xli, 59.

[8] Deut. med. Woch., 1906, xxxvi, 1444.

Rossel advises the use of freshly made tincture of guaiac. or, if an old tincture is used, this should be tested with a weak blood solution. Schumm also advises fresh guaiac. I have used old guaiac and found it satisfactory for a period extending over some weeks. The turpentine, of course, should be old and should not have stood in sunlight, and before using as a testing solution its reliability and maturity should be first tried on a bloody stool or on a blood mixture.

Aloin Test. Rossel does not lay much stress on disturbing factors, except fat. A fresh solution of aloin should always be made, as in time it assumes the red color seen with blood. There seems to be a difference of opinion between observers as to the value of this test. Rossel, Boas, and v. Koziezkowsky prefer it to the guaiac test, while Schloss seems to think there is nothing to choose between them. Schumm agrees with him and supports him in his view that both should be used as controls.

Benzidin Test. Owing to its simplicity, this test will doubtless find its way into every laboratory, but in using it several precautions must be borne in mind.

1. The benzidin must be pure, and only Merck's benzidin puriss. should be used. This was found by Schumm and Westphal to be ten times as active as the benzidin puriss. of other firms.

2. The same authors claim that a positive reaction depends on the relative amount of the individual agents. An excess of H_2O_2 can cause a disappearance of the green color, consequently, if too much H_2O_2 is used in making the test no green color appears. Therefore, the H_2O_2 should not be stronger than 3 per cent., and but 2 c.c. should be used.

3. The following substances were found by various authors to give a positive reaction with benzidin: Oxydizing ferments, animal as as well as vegetable (readily destroyed by boiling); iron salts, pus, saliva, nasal secretion, bowel detritus, and mucus give a more or less positive reaction; potassium iodide, pure animal charcoal, metallic iron, platinum, and copper in all forms. (One should be careful of the test tubes used for sugar reactions.[6])

Schlesinger and Holst made a comparative study of the aloin, guaiac, and benzidin tests, in the endeavor to find a simple test for occult blood in the feces. They used three modifications of the guaiac test, including Schumm's improvement, and in feces from persons on ordinary meat diet all three tests, the aloin, guaiac, and benzidin were found to be positive. On small amounts of meat (test diet) the aloin and guaiac tests were negative, excepting Schumm's modification of the latter, which was positive, as was also the benzidin test. In meat-free diet, all tests by all methods were negative.

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[6] Ztschr. f. phys. Chem., 1905, xlvi, 510.

In using the benzidin test as modified by Schumm, they did not observe a negative reaction. In two long meat-free experimental series made on themselves, the guaiac and aloin tests were negative forty-two to sixty-five hours after the last stool from a meat diet, while the benzidin test (Schumm) was positive on the eighth, even on the tenth day. As it is impossible to continue a meat-free diet for ten days, the Schumm benzidin test is hardly to be recommended. Schumm himself said the test was too "subtile," and hesitated about advising its use, as did Boas.

As regards the guaiac test, Schlesinger and Holst both consider Schumm's modification a refinement and, if made according to his directions, it is much more sensitive than aloin, whereas, if done in the ordinary way, aloin is the more sensitive, as the guaiac test is often masked by an olive-green color. When many feces are to be examined, Schumm's method takes up too much time to be of much service as a routine test. Schlesinger and Holst recommend the benzidin test in the above described method for the following reasons:

1. It is simpler than any other method.
2. It is sharper than the aloin and guaiac tests, but is, nevertheless, applicable for practical use, which the guaiac test of Schumm is not.
3. By boiling, all disturbing factors, such as ferments, are destroyed.
4. We have a control of the reagents, making the test more sure.

Schlesinger and Holst made experiments with weighed portions of blood-free stools and bloody stools, and found that 0.25 gram of tarry stools plus 5 grams of blood-free stools gave a positive aloin and guaiac test. With 0.05 gram + 4 grams the aloin and guaiac tests were negative, while 0.05 gram tarry stools + 20 grams blood-free stools gave the benzidin reaction (green). They made observations on themselves, taking meat-free diet until the stools remained negative for blood for some days, then, at luncheon, with a cup of tea and sugar, they took a weighed portion of fresh blood, 0.5 gram, obtained by pricking their fingers. On the third day, in one instance, there was a positive benzidin reaction, while, with Schumm's modification of the Weber test there was only a green color, which Schumm regards as negative. The examination of the other stool was negative, perhaps due to a disturbance of the intestinal function evidenced by fermentation and subjective symptoms. It is thus seen that ten drops of blood are sufficient to give a positive benzidin reaction, while, in comparison, Rossel's test first shows a reaction with 10 c.c. blood (after 5 c.c. generally negative), and Schumm's modification of the guaiac test a positive result with 2 c.c.

The afore-mentioned authors, together with Oettinger,[10] and Oettinger and Girault,[11] have used the test with various degrees of satisfaction; the rosy accounts of its partisans were so encouraging aging that I was led to make a comparative study of a number of feces, in order to learn what might be expected from this much vaunted and much disputed test.

I found, in the first place, that taking a piece of feces the size of a pea and mixing it in a test tube with water (as advised by Schlesinger and Holst) was not practicable; moreover, it did not seem reasonable to examine such an isolated portion of feces. If the blood is not equally distributed throughout the stool, the chances of selecting a portion of feces containing blood becomes questionable. I preferred to grind the feces with distilled water and to examine a boiled portion of this.[12]

According to Schumm, the first advantage claimed by Schlesinger and Holst, namely, simplicity, is sacrificed by boiling. The time necessary to bring the feces to a boil is insignificant, at the most fifteen to thirty seconds, and I fail to see how this manipulation interferes with the simplicity of the method. Schumm further contends that, while boiling destroys the oxydizing ferments it does not affect the inorganic substances, such as potassium iodide and animal charcoal, which substances react to the benzidin test. So, in common with v. Koziczkowsky and Schlesinger and Holst, he recommends the use of vegetable charcoal to mark off the feces. Schlesinger and Holst,[13] in a reply to this article, accept Schumm's suggestion about grinding up feces in a mortar, instead of selecting an isolated piece, and mention that in using the benzidin test potassium iodide and animal charcoal are always previously eliminated.

In the present series of observations no attempt was made to eliminate meat from the diet, as the primary object was to compare the relative values of the three tests, and not to make a clinical study of diseases from the viewpoint of the presence or absence of occult bleeding. The tests used in this series were the guaiac, the aloin, and the benzidin tests. For the first two tests a portion of the feces was first ground up with distilled water in a glass mortar, a portion of this put into a separating funnel and shaken with ether to extract the fat. By using repeated small portions of ether I rarely met with any difficulty from emulsion formations. Did this occur, the addition of more ether invariably caused the complete separation. The ether and feces were poured into a large, wide-necked test tube, and the ether then poured into a bottle for waste ether, to be redistilled later.[14] The remaining fat-free feces was shaken with equal parts of glacial acetic acid; then 10 c.c. ether was added and the mixture again shaken, and allowed to stand until the ethereal extracted had separated from the fecal residue. The acetic acid ether extract is used for the aloin and the guaiac tests.

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[10] Jour. des pratic., 1906, 151.

[11] La semaine méd., 1906, 325.

[12] Since beginning this research, Schumm, Munch. med. Woch., 1907, has published a paper in which he advises the same procedure.

[13] Munch. med. Woch., 1907, 160.

A few cubic centimeters of the extract are poured into one test tube and a few cubic centimeters of ethereal extract of guaiac are added, then an equal quantity of H_2O_2 (3 per cent.), and the whole shaken gently. Only the blue color of the ether was regarded as indicating blood, other colors being considered as negative.

Into another test tube are poured a few cubic centimeters of the glacial acetic acid-ether extract and 2 to 3 c.c. of the aloin solution is added. (This solution must be made fresh each time by dissolving as much aloin as will go on the end of a knife in about one-third of a test tube of 70 per cent. alcohol.) To this 2 to 3 c.c. of ozonized oil of turpentine are added and the test tube gently inverted several times. In the presence of blood the lower layer of aloin becomes a cherry-red color, but if the color does not appear in from five to fifteen minutes it should be considered as a negative test, as often by that time the aloin itself gradually assumes the same cherry-red color. The above two methods are those used by Steele and Butt,[15] and have been found to be most satisfactory.

Benzidin Test. For this purpose I recommend the following procedure: A small portion of the feces solution, obtained by grinding up the feces with water, is poured into a test tube and boiled. While this is cooling, a concentrated solution of benzidin is made by dissolving as much benzidin as is contained in a very small spoon spatula in about 3 to 5 c.c. of glacial acetic acid. A few drops (1 c.c.) are poured into a test tube and about 3 to 10 drops of this boiled feces solution further diluted with equal parts of water, are added and the two shaken. To this are now added 1 to 3 c.c. H_2O_2 (3 per cent.) and shaken, when, in the presence of blood, a deep, dirty green or a beautiful deep blue color appears, changing in the course of time to a peculiar dusty violet color. Only the green or blue color is to be considered positive. I have used larger portions of normal feces with negative results, but the importance of using small quantities of the reagent has been pointed out.

In the series of 100 feces the guaiac, aloin, and benzidin tests were carried out, in addition to the routine feces examination. I thought at one time in the study that the composition of the feces might play an interfering role in the tests, but I found the tests positive or negative, as the case might be, irrespective of reaction, bile pigments, fat, vegetable fibers, and very often meat. Of the

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[14] It was found to be more convenient to pour the ether from the large test tube, than from a separating funnel, as in the latter method it is almost impossible to remove more than half of the ether.

[15] Amer. Jour. Med. Sci., July, 1905.

100 cases some have been examined a number of times, and in all I have studied 129 feces. In order to avoid long tables of diet, microscopic and chemical findings, and diagnoses, I shall confine myself simply to a tabulation of the comparative results given by the three tests.

Aloin, guaiac, and benzidin positive	28	times.
Aloin, guaiac, and benzidin negative	82	"
Aloin positive, guaiac and benzidin negative	0	"
Guaiac positive, aloin and benzidin negative	0	"
Benzidin and guaiac positive, aloin negative	5	"
Benzidin positive, aloin and guaiac negative	2	"
Benzidin and aloin positive, guiaac negative	2	"
Benzidin and guaiac positive, aloin?	3	"
Benzidin and aloin positive, guaiac?	1	"
Benzidin and guaiac negative, aloin?	2	"
Benzidin and aloin negative, guaiac?	1	"
Aloin and guaiac negative, benzidin?	1	"
Aloin, benzidin, and guaiac?	2	"

In all, the three tests agreed 110 times out of 129 stools examined, whence I have no hesitancy in proclaiming the benzidin test an important addition to our laboratory equipment. From these studies I should advise first a test of the feces with benzidin; if negative we may be sure of the absence of blood, and we need go no further. If, on the other hand, the test is positive, the aloin and guaiac tests should be made, and if these are negative, occult blood may be regarded as being absent. A substantiation of the benzidin test by either the aloin or the guaiac test is to be regarded as indicating the presence of blood. Hence, a positive benzidin test shows nothing unless corroborated by one or both of the other tests; a negative test, however, is proof positive of the absence of occult bleeding. (In none of the cases was the benzidin test negative and the other tests positive.)

The clinical importance of a positive or a negative blood test is well known, has been dilated upon by various writers, including Steele, who made the first observations in the United States. At present I can add nothing to what has been already said.

I have also tried the benzidin test on gastric contents and the urine. Meanwhile, Schlesinger and Holst have reported their observations on the applicability of the test in detecting occult blood in the urine. For this they advise 10 c.c. urine + 0.5 to 1 c.c. of glacial acetic acid, invert a couple of times, then add one-third volume of ether and shake well. After standing a short time one can make a sharp separation by the addition of 5 to 10 drops of absolute alcohol and gentle shaking. If no separating funnel has been used, the ether may be drawn off with a pipette, and this extract treated with benzidin and H_2O_2 in the usual way, a green to a blue color indicating blood. They claim that the benzidin test is twenty times sharper than Heller's, and five times as sharp as the guaiac or the aloin tests, and they prefer it in testing urine for blood.

CONCLUSIONS. The benzidin test is to be recommended unreservedly for feces, gastric, and urine work. The objection to its extreme sensitiveness is invalid, as a control by the other tests may be made; while that very objection supports my assertion that if the benzidin test is negative the specimen may be considered free from blood. I advise checking a positive reaction with either the guaiac or the aloin test, preferably both.

2. The reasons given for advising the benzidin test as given by Schlesinger and Holst are sufficient, and need no further discussion.

3. Only the Merck benzidin puriss. should be used.

4. The quantities of the reagents must be carefully observed and the test tubes must be clean.

5. All disturbing factors must be first removed. By this I mean inorganic as well as organic. It appears to me as useless to examine a feces repeatedly for occult blood, making no effort to regulate the diet and medication, as it would be to examine the gastric contents chemically after a promiscuous meal, not taking into consideration the time, quantity, and quality of food ingested. Therefore, I advise, if the feces show a positive reaction for blood, that another stool be not examined until the diet and medication have been regulated. Only then can a fair idea of the clinical importance of the test be obtained.

FACTORS INFLUENCING THE EARLY DIAGNOSIS OF GASTRIC CARCINOMA.

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The advances of the last two decades in the laboratory and clinical diagnosis of gastric carcinoma, combined with the realization of operative possibilities, have been far-reaching, and it is safe to assume will acquire greater importance in the years to come. Following the early workers in the field of gastric analysis and deduction, the work of Billroth stands preëminent, and to the surgeons of our generation much credit is due for urging the necessity of early diagnosis if treatment is to be of avail. One has but to survey the operative results of the Mayos, Senn, Moynihan, Robson, Ochsner, and others to realize how much has been accomplished, and to regard with more hopeful optimism the outlook for the future. On the other hand, there probably will continue to be found much wisdom in the old adage, which refers to making haste slowly, for otherwise surgery will suffer the reaction which always accompanies the less careful diagnosis of cases in the enthusiasm of prospective cure.

The early diagnosis of gastric carcinoma, however, must remain the important factor leading to permanent aid or amelioration, and it is from this standpoint that I review the pertinent facts. No great amount of value need be placed upon the personal opinions expressed, except in so far as they bring out points for discussion, as to the methods in vogue, from the experience of those interested in the subject.

Carcinoma of the stomach claims a large percentage of those dying from malignancy in general. Judging from the census report of 1900, Dowd[1] states that there were 9000 deaths from carcinoma of the stomach in the United States during that year. This was 65 per cent. more than from appendicitis. McConnell[2] has shown that in the registration area (census 1900) there were 17,296 cancer deaths during that year, of which 4220 were due to cancer of the stomach (24.4 per cent.). Bland[3] has recently stated that typhoid fever has been endemic and epidemic in Philadelphia during the past twenty-five years During that time scarcely a winter or spring has passed without the city suffering a more or less severe epidemic, and 15,407 persons, including children, died of typhoid fever, while 15,379 died of cancer. In other words, in twenty-five years only twenty-eight more persons, including children, died from typhoid fever than from malignancy in a community where typhoid fever has been unusually prevalent. Cancer of the stomach claimed 37 per cent. of the total number. The comparative immunity of the colored race seems well established; 35.4 times as many in whites as in the colored race (according to McConnell's figures).

One may assume several possibilities in attempting to explain the frequency of carcinoma of the stomach as compared with other parts of the alimentary tract, and the theories, leaving out the possibility of its parasitic etiology as yet unsettled, are speculatively attractive, even if not contributory to definite fact.

Not the least of these has reference to the kind of food and misuse of the organ by practically all the higher animals. The lower animals rarely suffer from gastric ulcer or malignant new growth.

What connection the preponderance of uncooked articles of food may have upon the question would be, indeed, hard to say. The analogy is, perhaps, not a fair one, for, if carried further, the relative immunity of animals to certain other affections common to man justifies in some of us, at least, the belief that such comparisons are not parallel. So this theory of preponderant raw food, with little cooked food in the form of meat, etc., like similar conceptions, is likely to bring one more into a study of fancy than fact, despite the use which has been made of the subject in certain quarters during the past few years.

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[1] Facts Concerning Cancer of Stomach, Med. Record, January 20, 1906.

[2] Cancer in United States, Jour. Amer. Med. Assoc., April 28, 1906.

[3] Study of Deaths of Cancer in Philadelphia from 1879–1904, Surg., Gynec., and Obstet., November, 1906.

Our friends the vegetarians have, perhaps, after all established a partial truth, but without justifying their contentions, there can be no question that a simple dietary of cooked and uncooked food such as bread, pressed wheat bread, cereals, milk, eggs, and fats rich enough in caloric value and essential food ingredients, has much to do with the prophylaxis of gastric disease and abuse which a vast majority of individuals give to the stomach. The chow-chow, pickles, bizarre salads, hot breads, hot pies, etc., were assuredly the invention of a Mephisto with perfectly functionating gastric secretions.

This abuse of the gastric function concerns faulty mastication, over-eating, the pernicious habit of washing down the food with quantities of ice-water (a habit distinctively American), malnutrition, with resultant anemia from deficient quantity and quality in the case of the poor, and lowered resistance generally. In women other factors are found aside from the necessity of child-bearing, and the many times evident lack of care following delivery.

Prolapsed viscera, and general lack of tone, perhaps with atony and gastric dilatation, and, in delicate women, menorrhagia with secondary anemia, predispose to functional secretory disturbance, which paves the way in many cases to definite alteration of structure in the way of latent ulcer and glandular repair hyperplasias, which may, unfortunately, not stop at the completion of the repair stage; at a later time, they may undergo so-called malignant transformation. These considerations refer in many cases to generally mild, obscure dy peptic troubles with a moderate grade of ptosis or dilatation, extending over years and covered in the phrase "precancer stage" of Murphy, Graham,[4] and others. Many clinicians are of the opinion that a majority of the symptoms are of more rapid onset in gastric carcinoma; still, the latency is marked in a fairly large percentage of cases.

In the history, it is difficult to satisfy one's self as to the influence of heredity (16 per cent. of cases, Dieulafoy;[5] 7 per cent. of all cases, Musser[6]) as compared with other factors mentioned above, general dyspeptic symptoms, latent and frank ulcer, general nutrition, and habits. The belief is expressed that a history of malignancy in some one relative has slight suggestive value only. The possibility of predisposition to malignant change may be considered present. and due regard given to it, when two relatives give a positive history. It is surprising, in considering any malady, how large a percentage of families, if careful data are secured, give a so-called cancerous history.

Loss of weight and the age of the patient have only relative importance in early diagnosis. They should, of course, be taken for what they seem worth, together with the general symptom complex. Many patients with chronic functional secretory disturbance are markedly emaciated and profoundly asthenic. If the diagnosis in such cases is not based upon gastric analysis, inflation, etc., and the patient receives guess-work therapy, the guess may as well be wrong as right; and the picture, when the patient presents himself at a later time, is likely to be one of pronounced cachexia.

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[4] Boston Med. and Surg. Jour., August 31, 1905.

[5] Manuel Pathologie Interne, ii, 190.

[6] Trans. Coll. Phys. Phila., Noevmber 13, 1905.

As regards age, although a vast majority of the patients are over fifty years, carcinoma can hardly be called rare under thirty-five. In a limited experience, I have twice seen the diagnosis confirmed—in one patient at twenty-seven, and the other at twenty-nine years.

Strange to say, gastric carcinoma in early life seems to run a comparatively slow course. Mathieu[7] has reported the case of a man of twenty-five years with symptoms for three years; Catrin, a case lasting two or three years, both quoted by George Dock,[8] who has described a woman, aged twenty-four years, with symptoms for over one year; another woman of twenty-one years, with symptoms for nine months, and tumor for six months before death; and a man of twenty years, with symptoms for one year.

Primary sarcoma and myoma in the 62 or 63 cases on record has usually occurred in young individuals of seventeen to twenty-five years. A recent article by Lecéne and Petit[9] mentions as the most common form a large tumor, which may be pedunculated, projecting into the abdominal cavity. It may produce no symptoms on the part of the stomach, merely suggesting an abdominal tumor, while again it may simulate carcinoma. Yates[10] has also reported 3 cases from Oehsner's clinic. He concludes that sarcoma is less rare than has been supposed.

Pre-existent ulcer, since the classical publications of Cruveilhier and Rokitansky, has been generally recognized as an important point in the history predisposing to carcinoma. Probably more stress has been laid upon this point than is generally warranted.

Dittrich of Prague, in 1848, reported 160 cases in which ulcer and carcinoma were associated 8 times; Lebert reported 100 cases preceded by ulcer in 9; Rosenheim reported ulcer and carcinoma in 6 cases in 100; Sonicksen, 14 in 100; Graham, in 135 cancer cases, found a history of ulcer in 60 per cent.

Carcinoma undoubtedly occurs in those areas of the stomach wall most frequently the site of ulcer: in order of frequency, the pylorus and the posterior wall of the lesser curvature. A smaller proportion originates at the cardia and the anterior and posterior walls of the greater curvature. Ulcer may be present for many years with few, if any, classical symptoms, until perforation, hemorrhage, or scar-tissue obstruction is evident. This is especially true if the acidity is low. Riegel's rule that ulcer is almost always associated with excess of acid, had been found misleading. Wagner[11] and many others in approximate confirmation have found that excessive HCl is present in less than half the cases of ulcer; on the contrary, probably 25 per cent. of all cases of ulcer have had excessive HCl secretion at some time.

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[7] La semaine médicale, 1895, 225.

[8] Cancer of the Stomach in Early Life, Amer. Jour. Med. Sci., June, 1897.

[9] Revue de gynécologie, viii, 6.

[10] Sarcoma and Myoma of the Stomach, Annals Surg., October, 1906.

It is strange, when one considers these facts, that primary carcinoma is not more common upon an ulcer base in the duodenum. Mayo and Franklin,[12] in an experience of 1112 duodenal and gastric operations, have only twice recognized primary carcinoma of the duodenum, and only once developing upon the base of a duodenal ulcer. Still, to quote Dr. Mayo,[13] "ulcer of the duodenum is nearly as common as in the whole of the stomach."

SYMPTOMS. Simple dyspeptic disorders, anorexia, flatulency, and general diffuse abdominal discomfort at the height of digestion, four or five hours after meals, rather than acute local distress soon after eating, seem to be the important early subjective symptoms in most cases. Boas[14] is of the opinion that the symptoms are vague and indefinite in their onset, as a rule.

Vomiting can hardly be called an early symptom. It may be present in a rapidly-growing medullary carcinoma involving the body of the organ or an ulcus carcinomatosa involving the pylorus and leading to early obstruction.

Hemorrhage occurs late, as a rule, except in the cases of malignant change engrafted more or less rapidly upon simple or multiple erosions. The cell character of the neoplasm has something to do with the possibility of hemorrhage, the early tendency to ulceration in the soft encephaloid varieties, and the tardiness, if not complete absence, of hemorrhage in the more densely-growing scirrhus being important factors for consideration.

Pain. In early cases pain is not marked. When present it is usually comparatively late in the disease and is constant, of a dull, boring, heavy character, differing from the periodic or paroxysmal sharp pain of ulcer. Pain as an early symptom of ulcer, with or without associated hyperchlorhydria, is rarely absent, while in carcinoma not involving the pylorus, the patient, at least in a fair percentage of cases, is free from actual pain during the entire course of the disease.

Tumor. When a palpable tumor is present upon inflation, the patient can hardly be considered a subject for early diagnosis. On the other hand, a considerable tumor may be present involving the lesser curvature under the edge of the liver, and give little palpatory evidence even with the advantage of a thin-walled subject. Again, a tumor of the lesser curvature on posterior wall, may be pushed backward upon inflation of the organ and not be palpable.

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[11] Archiv f. Verdauungskrankheiten, xi, No. 1.

[12] Brit. Med. Jour., November 10, 1906.

[13] Jour. Amer. Med. Assoc., September 22, 1906.

[14] Diseases Digest. System. Edit. by Billings (Appleton, 1906).

When palpation is practised after inflation, the possibility should be remembered of the spleen being drawn downward by means of the gastrosplenic ligament, admitting ready palpation and possibly mistake as to the origin of the mass. Retroperitoneal masses may occasionally simulate a growth involving the stomach. Upon one occasion, recalled at the present time, a mass seemingly involving the stomach was shown, upon exploratory incision, to be a displaced kidney.

Likewise, upon another occasion, in an asthenic and cachectic patient, aged sixty-nine years, sick one year with dyspepsia, first examination revealed absence of free HCl, the presence of long bacilli, and lactic acid, together with a readily-palpable mass the size of a small orange in the upper epigastric region, which moved with deep inspiration and appeared to be connected with the stomach. A blood examination, made a few hours later, showed the condition to be one of pernicious anemia with 817,000 red cells. Upon free purgation, the mass disappeared. It was probably due to a distended transverse colon.

Chesnel[15] has mentioned cases of latent carcinoma with no hemorrhage, tumor, or vomiting. I have twice seen at autopsy in elderly individuals, scirrhus carcinoma involving the walls diffusely, but not the pylorus, in patients dying from intercurrent pneumonia, who gave no symptoms except loss of appetite and anemia in the few weeks under observation before death. Stevens[16] has described a case of extensive carcinoma of stomach, liver, and thoracic duct of seventeen months' duration in which there was entire absence of gastric symptoms until the very latest stages. Pitt[17] has also described 17 cases of gastric carcinoma giving symptoms, for the most part, unconnected with the organ (ascites; pleural effusion, 7 cases; matted intestine, 3 cases; obstruction, 2 cases; abdominal suppuration, 2 cases; anemia, 2 cases).

A priori, one would think gastric sufferers would present themselves to the physician earlier than in many other conditions not interfering so much with their feeling of well-being. If this condition of affairs prevailed, it would undoubtedly be of much assistance in the way of early observation of suspected malignant change. Figures, however, in my own cases at least do not show this to be so. I have averaged the length of time symptoms were present in 50 gastric cases before the patient presented himself for treatment and before an analysis was made. The average was about five months. Some of these had been treated before, but no analysis of the gastric contents after a test meal had been made.

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[15] Cancer latent de l'estomac. Th. de Paris, 1877

[16] Brit. Med. Jour., May 29, 1905

[17] Practitioner, London, April, 1905

The suspicion of malignancy in most instances rests upon the ordinary physical examination; but before one can safely exclude malignant change, resort to other useful adjuncts should be secured. In practice, perhaps the majority of physicians do not use the test-meal and the stomach tube. So much has been written relative to the subject, whether one should use one analytical indicator or another, the variety of test meals, the lack of familiarity with sarcines, yeasts, bacilli, lactic acid tests, etc.; in general, the subject has appeared to possess real technical difficulties. For ordinary clinical work much knowledge may be obtained with very little labor, in fact less time than one would consume in making a complete urinary examination.

One may obtain practically all the facts essential to a tentative diagnosis without the use of complicated apparatus. A little practice in reading indicator reactions, in knowing the Kelling, Uffelmann, or Knapp[18] floating ether tests for lactic acid, the microscopic appearance of normal and abnormal gastric elements, and the occult blood tests, would avail much toward the recognition of facts available by no other means.

Too much is expected, as a rule, from one or two examinations. That serious organic disease can be excluded by one or more perfunctory tests is akin to the fallacy of attempting to exclude chronic interstitial nephritis by one or two urinary examinations. The urine may show no albumin for weeks in this most fatal form, while the fact that free HCl is present in the chyme, does not exclude gastric carcinoma or other organic change.

The supposed difficulty on the part of patients in taking the tube is more imaginary than real. A seemingly small chronic ulcer should not contra-indicate its use (symptoms pointing to more or less erosion with evident hemorrhage should probably be contra-indicative). Little trouble need be anticipated in chronic ulcer cases if the procedure is adopted when the tube is half way down the œsophagus of allowing a pint of water to flow down into the stomach in advance of it. Valuable facts may sometimes be secured from these washings.

Complicated analyses, such as it has been my privilege to see occasionally from sanitaria, etc., are rarely necessary. A simplified method of examination suffices: first with an empty stomach in the morning, then with a test meal of one shredded wheat biscuit with 300 c.c. of water removed in fifty minutes (modified Ewald-Boas), and within the next day or two, in order to test the digestive ability under a more tasteful stimulus, the same meal, to which has been added one-quarter pound of chopped beef, broiled, and seasoned slightly, to be removed in three to three and one-half hours (modified Riegel, or modified Germain-See's). This regime, repeated as often as necessary; inflation of the stomach with an ordinary hand-bulb attached to the end of the tube; and, in thin wall subjects, gastric illumination, will be found quite sufficient.

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[18] Chyme Analysis, Med. News, New York, November 14, 1903; also Amer. Med., May 7, 1904.

Despite the controversy concerning the reaction of organic acids to dimethylamido-azobenzol in the test for free HCl, it has been found satisfactory in the vast majority of cases. When organic acid and stagnation are marked, either due to hypochlorhydria or from dilatation secondary to benign obstruction at the outlet (muscular hypertrophy, pyloric spasm, perigastric adhesions with deficient motor power, the scar of an old ulcer) or, malignant constriction, it is better to titrate for free HCl with the dimethyl first; then titrate a separate specimen with a saturated alcoholic solution of tropeolin 00, and subtract the one from the other, the difference being the amount of organic acid present; while the tropeolin titration may be taken as indicative of the amount of free HCl present.

Combined HCl is rarely absent when free HCl is present, and the estimation of the former is rarely necessary in ordinary clinical work. When free HCl is present pepsin or pepsinogen will rarely be found absent. However, the convenient Mette test or egg disk consumes very little time and should be used.

As regards evidence of stagnation in the fasting stomach contents, Ackerman and Gompertz[19] have found that lactic acid and sarcines are supplanted by Oppler-Boas bacilli, and, when abundant, are of much more diagnostic import as indicative of malignant stenosis at the outlet than the finding of sarcines in benign obstruction. With Ziegler's[20] work on the microscopic stagnation of stomach contents as a sign of carcinoma in the lesser curvature, I have had no experience. He contends that the persistence of lactic acid bacilli *after lavage* points to the great probability of carcinoma. In noncancerous cases the stagnation is microscopic or entirely absent, and the lactic acid bacilli are readily removed by one or two stomach washings, while in carcinoma their persistence is the important point, even though the motor function seems to be intact.

There can be no doubt that free HCl is absent, at least when most of the symptoms of malignancy are evident, in a large majority of cases (Wagner,[21] 90 per cent.; Howard,[22] 92 per cent.;). A certain number of cases not infrequently, however, show hyperacidity, especially when the growth is engrafted upon an ulcer, the so-called carcinomatosa.

Recently the very important fact has been brought out by Benjamin Moore[23] and confirmed by Palmer,[24] that there is a marked reduction in HCl secretion in patients with malignant growths, no matter where located in the body, and that this diminished secretion is not dependent upon the location of the growth in the stomach. It has been known for a long time that in the general malnutrition of malignant change involving any part of the body, or from any other cause, the stomach secretions are altered, and that this alteration many times manifests itself in greatly diminished HCl. The work of Moore and Palmer deserves especial emphasis in connection with this subject.

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[19] Diagnostic Value of the Microscopic Examination of the Fasting Stomach Contents, Med. Record, April 8, 1905.

[20] Zeit. f. klin. Med., lviii, 6.

[21] Archiv f. Verdauungskrankheiten, xi, 1.

[22] Ulcer of the Stomach and Duodenum, Amer. Jour. Med. Sci., December, 1904.

With Sahli's[25] *desmoid reaction* the results are often misleading. Its value, however, is unquestioned in certain cases. The reaction has occasionally been found positive when the secretion of gastric juice was greatly reduced, and again negative with the gastric functions normal.[26]

The detection of *occult blood* in the stools and stomach contents has proved to be of genuine diagnostic worth. The Adler technique (of benzidin in 90 per cent. alcohol, glacial acetic acid, and hydrogen dioxide) has been found absolutely reliable.[27, 28] It shows the presence of blood even in a dilution of 1 to 250,000. Friedenwald and Rosenthal[29] found blood present in practically every case of carcinoma of the stomach examined. To be reliable, no meat should be eaten for two or three days beforehand, and no raw vegetables. Hemorrhoids, the pulling of a tooth, bleeding from the nose or pharynx, occasional intestinal bleeding due to cirrhosis of the liver or tuberculous ulcers, should be carefully excluded.

Occult hemorrhages are never encountered in healthy individuals. Certain forms of cancer, such as the dense slow growing scirrhus, may not be accompanied by occult hemorrhages, but these forms are comparatively rare. With the sensitive Adler technique, Oettinger and Girsuit[30] have found traces of blood frequently in the stools in cases of pure hyperchlorhydria.

Tissue Fragments and Albumin Digestion. As a rule, time spent searching for tissue fragments for microscopic examination in the stomach contents can be better used in other ways. Distinct tumor particles have been occasionally found, but, as a rule, avail one little in the general run of cases. The work of Rosenburg[31] relative to the amount of nitrogenized residue formed from the albumin in the cancerous stomach will hardly prove of value in early diagnosis. Normal variations, and especially the variations in the different gastric neuroses, will make the findings uncertain and of questionable utility.

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[23] Proc. Royal Soc., 1905, lxxvi, 138, and Biochem. Jour., 1906, i, 274; Jour. Amer. Med. Assoc., March 2, 1907.

[24] Biochem. Jour., 1906, i, 389.

[25] Corresp.-blatt. f. Schweitzer Aerzte, 1905, xxxv, No. 8-9.

[26] Deut. med. Woch., xxxiii, 1906, No. 22.

[27] La semaine médicale, 1906, xxvi, No. 28.

[28] Deut. med. Woch., 1906, xxxii, No. 36.

[29] Meet. Chirurgical Fac., Maryland, Baltimore, April 24, 1906; also New York Med. Jour., August 11, 1906.

[30] La semaine médicale, 1906, xxxvi, No. 28.

[31] Ztschr. f. klin. Med., 1899, lvi.

Pyloric Stenosis. Sahli[32] is of the opinion that pyloric stenosis is diagnosticated too frequently, when in reality a somewhat delayed emptying of the stomach may be nothing more than a functional weakness of von Mering's reflex, or slight insufficient motor power. He feels that we are not justified in diagnosticating offhand a pyloric stenosis from the presence in the gastric contents of remains of food taken the previous day.

Gastric Rigidity and Motility. Penzoldt's Iodine Test. The stomach motility in a carcinoma not involving the pylorus may be quite normal. Gastric rigidity has seemed to offer little of value for early diagnosis. The iodine test depends probably as much upon the stomach motility as upon the absorbability of the substance by the mucous membrane. Von Mering[33] has found that potassium iodide is not absorbed at all by the stomach in the dog even after two or three hours, and that the potassium iodide is absorbed almost solely by the intestine.

Enlargement of the Supraclavicular Glands. Enlargement of the glands near the attachment of the sternomastoid muscle on the left side especially has been considered a sign of a certain value in gastric carcinoma. Such enlargement may be difficult to tell in early cases from simple inflammatory resection due to other causes. It has been found present also in cases of ulcer of the stomach.[34]

Trousseau laid great stress upon an *obliterative phlebitis* as one of the early signs of gastric carcinoma. In fact it was the appearance of phlegmasia alba dolens which causes him to confirm the diagnosis of cancer of the stomach in his own case and to which he succumbed nine months later.

Elliott[35] is of the opinion that *a Gram-positive stool*, while in no way pathognomonic of carcinoma of the stomach, is still very suggestive of this condition, while a *Gram-negative stool* speaks decidedly against it. The field of study is an interesting one and should be more fully worked up.

Blood Examination. For early diagnosis the blood has no distinctive features in gastric carcinoma. After the process is firmly established the changes found in secondary anemia of moderate grade are usually present. The hemoglobin is reduced to 40 to 60 per cent., in late cases even to 20 to 25 per cent., while the color index remains below 1.0, differing in this respect from idiopathic pernicious anemia or aplastic anemia. The leukocytes, as a rule, are moderately increased, averaging from 10,000 to 15,000, and this is a very important point in diagnosis. The leukocytosis is of the polymorphonuclear variety.

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[32] Diag. Methods, 114.

[33] Klin. Jahrb., 1899, vii.

[34] Soc. méd. des Hôpit., January 25, 1895. (Quoted by Dieulafoy.)

[35] Chicago Med. Recorder, June, 1906.

Exploratory Laparotomy. In a given case, in which simple dilatation is excluded, if evidence of stagnation is present, if under the various test-meals it is found that seemingly permanent secretory alterations have taken place in the way of deficient HCl or excessive organic acids, with the finding of yeasts and long bacilli, and in which the secretion and stagnation are not improved by appropriate medical and dietetic treatment, it is safe to assume that organic disturbance is present. It is not unusual, however, to see a gain of from 8 to 12 pounds shortly after beginning treatment in cancer cases; this, although confusing, should not mislead one in the constant search for evidence of organic disease. Such findings, with or without vomiting, with or without blood, with or without tumor or emaciation, in an individual over forty years, should make one extremely suspicious. Prolonged attempts, to be sure of one's ground from the laboratory standpoint, will lead many times to dangerous delay. One of the apparent reasons why these patients come to the surgeon so late is because of the procrastination so many times evident. Dr. Mayo has found in 313 operative cases that only 26 per cent. were early enough to permit of radical extirpation. These facts should make us more anxious to give such sufferers the benefit of competent surgery, and the whole matter hinges alone upon early diagnosis. "A suspicion of cancer of the stomach, which cannot be disproved by known methods within a short time, should lead to exploration" (Dr. Mayo). This is especially true if there is a chronic ulcer element present in the history, with return of dyspeptic symptoms not amenable to treatment in a few weeks (seven or eight weeks). The presence of icterus, of enlarged clavicular glands, prominent subcutaneous veins over the abdomen, ascites, or more or less constant pain, lessens much the chance of aid, although Robson, Sheldon, and others have mentioned cases supposed to be clearly inoperable, in which recovery has ensued after the temporary drainage expedient, gastro-enterostomy. Such a course of events make it evident that malignancy was not present in the beginning. These border-line cases are difficult of interpretation at best, but when doubt exists, exploration in competent hands should be urged, with the hope that the circumstances after the abdomen is opened will justify procedures leading to permanent aid or amelioration.

DIFFUSE DILATATION OF THE ŒSOPHAGUS WITHOUT ANATOMICAL STENOSIS, WITH THE REPORT OF A CASE DUE TO CHRONIC CARDIOSPASM.

By William Lerche, M.D.,
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In 1900 Neuman[1] was able to collect from the literature 70 cases of diffuse dilatation of the œsophagus without anatomical stenosis. In 1904, Mikulicz[2] estimated that at least 100 cases should be collected. The first case on record seems to be that of Purton,[3] in 1821. Huss,[4] in 1842, reported a case in which he made the diagnosis of dilatation of the œsophagus and stenosis of the cardia. At the postmortem he found a dilatation but no stenosis, and he considered spasm of the cardia the cause of the dilatation. Most of the earlier cases were first recognized on the postmortem table, but during the last twenty years a number of cases have been diagnosticated during life.

The early work of Mikulicz and Meltzer has done much to throw light upon the etiology and pathology of this peculiar condition, but as yet it is somewhat obscure. There are several factors to be considered in the etiology. Fleiner,[5] who looks upon the dilatation as congenital and calls it *antestomach* (vormagen) following Luschka, has adopted Mehnert's[6] enteromere theory to explain this. The primary foregut from which the œsophagus is derived is segmented like a rosary and the musculature may be looked upon as consisting of a number of segments, which Mehnert calls enteromeres, separated from one another by a narrow zone or constriction. Each segment corresponds to a vertebra and each narrow zone to an intervertebral disk. There are twelve segments and thirteen narrow zones or constrictions. One segment is below the diaphragm and the rest are above. Fleiner thinks that from an anomaly in development, one or more of these enteromeres may remain, and the muscular fibers, being weak, will give way. Luschka has described a subdiaphragmatic dilatation of the œsophagus, found postmortem, above the cardia, which he calls *antrum cardiacum*. Cases of congenital dilatation seem to be rare. One case of Fleiner's was found accidentally postmortem and there was no history obtained pointing to œsophageal affection.

Zenker[7] mentions a case of a seven months' child, who died when seven days old, in which was found a spherical ectasia, sharply bounded above and below (next to the cardia) by a very thick, bluish-red wall, in consequence of hemorrhagic infiltration. Sievers[8] reports a case with difficulty of deglutition since earliest childhood, which he considers congenital.

Gottstein[9] thinks that in Case No. 15 in his article there was a congenital dilatation (vormagen), in which catarrhal conditions had come on with consequent cardiospasm. In Case No. 87 in the same article, Gottstein found a subdiaphragmatic dilatation of the œsophagus (antrum cardiacum), spasm of the cardia, and an ulcer on the cardia which later proved to be carcinomatous. Both of these last-named cases were examined with the œsophagoscope.

Primary atony of the muscular coat of the œsophagus as the cause of dilatation, with subsequent cardiospasm, has been described by Rosenheim and Netter. Netter[10] first called attention to this, but Rosenheim has particularly brought out the importance of this as an etiological factor, chiefly through his œsophagoscopic observations. Rosenheim,[11, 12, 13] describes cases of simple atony of the œsophageal walls, in which the solid food passes very slowly through the tube, causing discomfort, pressure, and pain, but in which there is no stenosis. This in time, however, may give rise to dilatation and cardiospasm. In these cases there is no hindrance to the passage of fluids, and the œsophageal bougie does not meet any obstacle. On examination through the œsophagoscope, the rosette-like folding of the mucous membrane of the cardia is not seen as in cases of cardiospasm. Rosenheim says: To be sure, disturbances of the innervation are at the bottom of this disease, but they are mostly local, and remain local, although general neurosis may be a predisposing momentum, or come on later. Any disturbance in the function of the nerve-muscle apparatus of the œsophagus, especially the cardia, any weakening of the peristalsis, any injury to the cardia, may be followed by ectasia; and what commenced as a simple functional neurosis may end as an irreparable pathological and anatomical process. Rosenheim has also described cases of primary spasm of the cardia without any discoverable lesion. He says that any pathological process in the œsophagus may be accompanied by spasm

Krauss (cited. by Lossen[14]) says that there may be simultaneous atony of the œsophagus and spasm of the cardia, and bases his opinion upon finding degeneration in certain branches of the vagus in a case of dilatation of the œsophagus with cardiospasm.

Zenker considered inflammation of the mucosa as a possible cause of ectasis, and refers to the case of Stern's,[15] in which the mucosa was found greatly hypertrophied, but not the muscular layer.

Martin[16] thinks that inflammation is the important etiological factor in the cardiospasm, and he supports his opinion with the case he reports in which œsophagitis with much secretion of mucus was present from the onset. However, inflammation, ulcers, etc., may be present without giving rise to spasm. Gottstein has observed cases of œsophagitis without spasm and dilatation. Rosenheim mentions 3 cases of chronic œsophagitis with dysphagia, but without any spasm, diagnosticated by the aid of the œsophagoscope.

There was no hindrance to the passage of the sound into the stomach. He also says that there may be œsophagitis without dysphagia. Rosenheim thinks pharyngeal catarrh should also be taken into consideration in diseases of the œsophagus. Ulcer of the œsophagus may be present without giving rise to œsophageal symptoms. Thus, Tileston[17] reports 3 cases of peptic ulcer of the œsophagus, seen postmortem; in the first case there was no symptom referringto the œsophagus; in the second case there was difficulty of swallowing solid food the last few weeks before death; the third case had stomach trouble one year before and gastric pain and vomiting the last two weeks of life.

Mikulicz, and later Meltzer, described cases of dilatation of the œsophagus in which spasm of the cardia was the primary cause. Mikulicz, as early as 1882, according to Gottstein, had observed 6 or 7 such cases and came to this conclusion mainly through the use of the œsophagoscope. Mikulicz gave the condition the name of cardiospasm. As to the cause of the cardiospasm he does not say whether he thinks it due to primary neurosis, œsophagitis, or congenital defect. Meltzer,[18] in 1888, reported a case of this kind. Einhorn,[19] in 1888, reported a case in which he thought a lack of reflex relaxation or opening of the cardia was the cause. Leichtenstern,[20] in 1891, came to the conclusion (independently of Mikulicz) that the spasm of the cardia was the cause of the dilatation with hypertrophy of the œsophagus in a case upon which he made a postmortem two hours after death. Both nervi vagi and branches were examined microscopically and found normal. Kitt (cited by Gregersen[21]) states that dilatation of the œsophagus has been found in horses and sheep and that sarcosporidia were found in the muscle cells. In the majority of the older cases (17 collected by Zenker) that came to postmortem, the dilatation was accompanied by hypertrophy; subsequent cases of similar kind that have come to postmortem are those of Strümpell,[22] Leichtenstern, Rolleston,[23] Johnson,[24] Gregersen, Zinsser,[25] Sippy,[26] Kinnicutt,[27] and Strauss.[28]

The specimens examined postmortem usually reveal a dilatation of the lower two-thirds of the organ, the dilatation having the bottle shape, or is spindle-shaped. The mucous membrane is frequently found inflamed and at times ulcerations are present. The muscular coat, particularly the circular fibers, are found hypertrophied. The small cell infiltration may extend down to the muscular coat. The cardia is mostly found normal, sometimes narrower than usual. In some cases the œsophageal wall is found thinned throughout, or a part of the wall may be thinned and bulging.

According to Neumann, both sexes are about equally affected. Of 41 cases, 22 were in men and 19 in women. Of Rosenheim's 9 cases, all were in men. Most of the cases reported have been between the ages of twenty and forty, but a patient of Fleiner's was seven years old, and a patient of Rolleston's was eight years old.

Gottstein reports a case of acute cardiospasm without dilatation in a child four years old, examined with the œsophagoscope.

The etiology of dilatation of the œsophagus without anatomical stenosis is far from clear, but it seems to me, from a study of a number of cases reported, that the clinical picture varies, and that cases in which atony is primary to the dilatation and spasm, if possible, should be differentiated from the cases in which cardiospasm is primary, as the treatment and the prognosis differ. Gottstein has arranged the cases as follows: (A) Cases in which excessive extraphysiological spastic contractions of the musculature take place (hyperkinetic disturbances of motility. (B) Cases in which the contractibility of the musculature is weakened or is entirely lost (hypokinetic disturbances of motility). The hyperkinetic and hypokinetic disturbances are divided according to the etiology into (1) The idiopathic or those in which the organic lesion cannot be demonstrated, and (2) the secondary or symptomatic, that is, those developed in connection with disease causing anatomical alterations, as cancer.

(A) Hyperkinetic disturbances of motility.

Idiopathic.

1. Œsophagospasm, when the spasm involves the œsophagus proper.
2. Cardiospasm, when the cardia is involved.

Cardiospasm—acute, chronic.

A definition of cardiospasm is given by Leichtenstern as a pathological exaggeration of a physiological phenomenon, in which, through an abnormal innervation of the cardia, a habitual, not permanent, spastic closure of the cardia takes place, which is an increase over the normal, lasts for a longer time, and especially takes place after the act of deglutition. Evidently, disturbances of the nervous apparatus of the cardia are at fault, but the question whether the condition is due to failure on the part of the inhibitory nerve fibers controlling the normal tonus of the cardia, or to irritation that has caused an increased tonus in the contracting fibers, may be difficult to determine.

Von Openchowsky[29] found that the vagus nerve contained contracting, as well as dilating fibers, and he demonstrated the brain centres for contraction and dilatation of the cardia. He also found ganglion cells in the serosa of the cardia which have to do with the function of dilatation. If the cardia is cut out of a living rabbit there will be seen rhythmic contractions. The cardia at rest is in a light tonic contraction, and, according to Sinnhuber,[30] the cardia-musculature is strengthened by the surrounding muscles of the diaphragm and the slanting insertion of the œsophagus into the stomach. Sauerbruch,[31] experimenting on dogs, found that the cardia contracted rhythmically synchronous with inspiration, and that the contractions were caused mainly by the musculature of the diaphragm. Mikulicz says that there is a valve-like shut-off on the stomach side of the cardia, and cites Gubarow and Kelling. H. Kronecker[32] and S. Meltzer,[33] in their experiments on the mechanism of deglutition, found that fluids were squirted into the œsophagus to the cardia by the action of the mylohyoid muscle, remaining there until the peristaltic wave followed, when they would go through the cardia. Solid food, after being forced into the œsophagus, would be moved forward by the peristaltic wave of the œsophageal walls. The mass would pass the cardia four to six seconds after the act of deglutition. Cannon and Moser[34] found that in man and horse liquids are propelled deep into the œsophagus at a rate of several feet a second by the rapid contraction of the mylohyoid muscles. Solids and semisolids are slowly carried through the entire œsophagus by peristalsis alone. Schreiber,[35] in a recent monograph, differs from Kronecker and Meltzer in regard to the muscles involved in the act of swallowing. Schreiber says that the mylohyoid muscle, when the mouth and nasopharynx is shut off, throws the food into the pharynx, where the constrictor media muscles and others press the bolus down into the œsophagus. He also says that the constrictor inferior antagonizes the swallowing muscles, as it holds the upper end of the œsophagus in a tonic closure; and that, when the food has passed into the œsophagus, the constrictor inferior closes after it. Schreiber found that fluids are not squirted down to the cardia, but are moved by peristalsis, like solid food. Mikulicz differs from Kronecker and Meltzer in regard to the passage of fluids. From experiments he found that fluids, after being pressed into the œsophagus, by their own weight, will open the cardia automatically without the aid of peristalsis. Mikulicz thinks that the ganglion cells in the cardia serosa have to do with the automatic opening on light pressure. Irritating fluids, as very hot or very cold, and irritating chemical fluids, make an exception to this, as they may cause an increased contraction of the cardia.

The œsophagus, on an average, is 25 cm. long, and, according to Mehnert and Mikulicz, is a straight tube. There are usually one or more physiological constrictions of the organ present, and the ones at the commencement and at the tracheal bifurcation and at the end are those most frequently found. Mehnert found in 50 per cent. of bodies that the œsophagus increases in width from above down, and he gives v. Hacker's measurements as 13 mm. diameter at the commencement, gradually increasing to 22 mm. in diameter above the cardia. Mikulicz found the diameter in the upper two-thirds to be 16 mm., in the lower one-third, somewhat narrower. The capacity of the organ varies from 40 c.c. to 80 c.c., but even 150 c.c. may be within physiological limit, according to Rumpel. The tube is closed in the neck part, but is open, and contains air in its passage through the chest (Mikulicz, Rosenheim,[36] and Chiari). According to Morosow,[37] the average distance from the incisor teeth to the commencement of the œsophagus in adults is 14.8 cm. The average distance from the incisor teeth to the cardia is, according to Rosenheim, 39.9 cm. in men and 37.3 cm in women. The position of the cardia changes with age; in the newborn it is found at a level with the eighth dorsal, and in old people may be as low as the twelfth dorsal vertebra, the average being between the ninth and eleventh. The cardia and œsophagus sink somewhat when the stomach is loaded. Mikulicz[38] and Schlippe[39] have made a number of observations on the intraœsophageal pressure. Mikulicz found that the pressure during rest is a trifle below the atmospheric pressure. By quiet inspiration the pressure is lowered to —9 cm. water pressure, and by forced inspiration to —20 cm. or below. On quiet expiration the pressure goes up to +10 cm. water pressure, and by forced expiration to +20. By coughing, the pressure may be 60, 80, or even 160 mm. mercury. On deglutition the pressure varies between 0.80 and 22 cm. water. The normal cardia opens easily without the aid of deglutition for fluids and gases going from the œsophagus into the stomach, but with difficulty in the reverse direction. The pressure necessary to open the cardia from the œsophageal side ordinarily amounts to a fraction of the pressure of a column of water filling the chest part of the œsophagus. When irritating fluids, as carbonized fluids or very hot or very cold fluids, are taken the pressure is higher.

If, now, the resistance of the cardia is increased, a part of the fluid swallowed will remain in the œsophagus. Suppose that, to effect the automatic opening of the cardia under normal conditions, a pressure of 12 cm. water pressure is necessary. In that case the balance of the fluid above the 12 cm. would flow into the stomach by its weight, leaving behind a 12 cm. column of fluid. This would be carried into the stomach by the pressure caused by the following act of deglutition, which corresponds to about 12 cm. water pressure. It the resistance of the cardia corresponds to 24 cm. water pressure, there will be left a column of 12 cm. at the end of the act of deglutition. If the resistance on the part of the cardia is still higher, only so much fluid will pass the cardia as is pressed down by the pharyngeal musculature. The œsophagus itself can only overcome the resistance of the cardia when it by energetic contraction can raise the pressure correspondingly. As long as the œsophagus has its normal dimensions the effect of the pressure is comparatively small, but when the organ becomes more dilated, so that the area upon which the pressure can be exerted is considerable, the dilatation increases more rapidly. The secondary changes in the œsophageal wall brought on by the stagnating ingesta may add to the increasing rapidity with which the dilatation takes place.

Mikulicz thought that the information gained by measuring the intra-œsophageal pressure in cases of dilatation would be of great value in diagnosis, as well as clear up the etiology. The pressure during deglutition would be an indicator of the condition of the contracting power of the muscular wall of the œsophagus. The apparatus used is very simple and consists of a rubber tube connected with a manometer. He reports 3 cases in which the pressure was estimated in this way. The following is the history of one of my cases:

Mr. H. B. E., aged twenty-seven years, unmarried, clerk, was seen January 17, 1907. Father well, mother nervous, brothers and sisters well. The patient had measles when a child and bronchial cough during the winter months from the age of ten to fifteen; otherwise his health was excellent. No specific history. He has always been in the habit of eating fast. He never had any throat trouble until the age of seventeen, when he had an attack of sore throat and swollen tonsils. During this attack, while at the dinner table, he was taken with a choking spell, turned blue in the face, and was not relieved until the food had come back. From now on he had difficulty in getting the food into the stomach, and in the course of two weeks the present condition was fully developed. Present complaints: when the patient has eaten about one-fourth of his meal he has a sensation of choking back of the larynx, and he is obliged to take a large quantity of water, 300 to 400 c.c., to force the food down. If he does not succeed in getting it down, all the food and the water come back. He repeats this three, four, or five times during the meal. Sometimes he feels that the food commences to pass into the stomach while he is drinking the water, at other times not. He always gulps up some of the water used to force the food down. At some meals nearly all the food and water come back, and he has to leave the table unsatisfied. If he drinks between meals, the water is always regurgitated. Very hot or very cold drinks increase the difficulty. He takes only two meals a day because he cannot get home to his midday meal, and he does not want to go through the performance in a public place. At times he will wake up in the night with what he calls a "contracting pain" to the right of the base of the ensiform process. If he then takes a drink of water and gulps it up again he feels relieved. Of late he has taken a small quantity of whisky and let it remain, with relief. He never found that any food or drink had run out through his mouth or nose during sleep. At times the "contracting pain" will come on on swallowing anything, solid or fluid.

During the first part of January of this year the patient was worse than usual, and he could only get very little food down for four or five days. Ever since this dysphagia commenced it has never left him, but at times he has had but little difficulty. He is particularly bad when he has a cold or when he has to work hard, or if he worries. In the first part of December, 1906, he was sick with ptomaine poisoning and tried to vomit, but he did not succeed. He had his tonsils removed years ago.

Examination: Slight catarrhal pharyngitis. Bloodvessels, heart, and lungs negative. On introducing a soft stomach tube it meets resistance at 15 cm. from the incisor teeth, and the patient has a choking spell; it is difficult for him to get his breath. After several attempts the tube was passed. There was evidently a spasm of the upper end of the œsophagus, which was noticed on every subsequent examination. The tube now passes readily down to 42 cm., when another obstacle is met with, and it is necessary to use a wire stylet in the tube. After gentle pressure is exerted for some time, the cardia suddenly gives way and the tube passes into the stomach. The stomach was inflated with air and found of normal outline. I found that the patient could drink 410 c.c. water and regurgitate it all after a few minutes. At one time he regurgitated 425 c.c.; the excess was mucus. This amount could be siphoned off from the œsophagus. After a test meal of toast and water, drawn off one hour later, I first obtained masses of bread enveloped in mucus from the œsophagus; when the tube was passed into the stomach by the aid of the stylet, the contents differed in appearance. The œsophageal contents was alkaline in reaction. The contents of the stomach showed normal acidity. On the introduction of the œsophageal bougie there was a violent spasm of the upper œsophagus and the bougie was passed with difficulty. Through the cardia it passed without hindrance. It was my intention to examine through the œsophagoscope, but I had to desist on account of the great irritability of the upper end of the œsophagus. For the same reason I could not use the double tube test of Rumpel to differentiate between dilatation and a deep-seated diverticulum. I used instead a modified Rumpel test suggested by Starck (cited by Kramer-Peterson[40]). The ordinary stomach tube was passed into the stomach and 750 c.c. of water poured in. The patient was then given 250 c.c. of milk to drink (with the tube in situ). The water was now allowed to flow off from the stomach and it returned clear. The tube was then pulled into the œsophagus and the milk was siphoned off. All the milk was recovered except 10 c.c. I now introduced with the stylet a stomach-tube to the end of which was attached a thin rubber bag covered with a silk bag, which, when distended, had a pear shape, and had a diameter of 3.75 cm. This was distended with water and could be moved up and down in the œsophagus for several centimeters. A larger size, 5 cm., was then used. I found that this could be moved only slightly, so I concluded that the dilatation was about 5 cm. in diameter. The swallowing sound was not heard at the ensiform process. After giving the patient 20 per cent. bismuth in thick oatmeal gruel, *x*-ray pictures were taken, but they were not satisfactory.

Diagnosis: Diffuse dilatation of the œsophagus, due to cardiospasm (and œsophagospasm). The diagnosis was based on the sudden onset with regurgitation of food and fluids; the fact that at times he had little difficulty in passing the food into the stomach, while at other times he could not get anything down at all; the hindrance to the passage of fluids from the onset; the peculiar sensation in passing the stomach tube with the stylet or the bougie; the sudden giving way of the cardia on gentle pressure; the ease with which the tube could be passed at times, and the difficulty encountered at others; the result of sounding with the pear-shaped rubber-silk sound; the capacity of the organ 425 c.c. The history would exclude cancer or other growth; the absence of a history of swallowing caustics, and the ease with which the large-sized bougie could pass the cardia, excluded benign stricture. The result with the modified Rumpel test excluded diverticulum, and so did the fact that the tube or bougie never entered a pocket; the resistance met was always at 42 cm. from the teeth, and it could always be overcome by gentle pressure without changing the position of the tube. There was never any food found in the œsophagus from the previous day's meal; the contents of the œsophagus was alkaline or neutral.

To exclude primary atony, the regurgitation of fluids from the start, the sudden onset, the promptness with which the patient could eject the contents of his œsophagus, aided. This latter fact, and the comparatively little pain in this case would rather lead one to think of dilatation with hypertrophy and with very little inflammation of the mucosa. That the inflammatory condition of the patient's throat and tonsils had been an exciting cause seemed quite reasonable. There must, however, have been an abnormally excitable condition of the nervous mechanism of the œsophagus and cardia present.

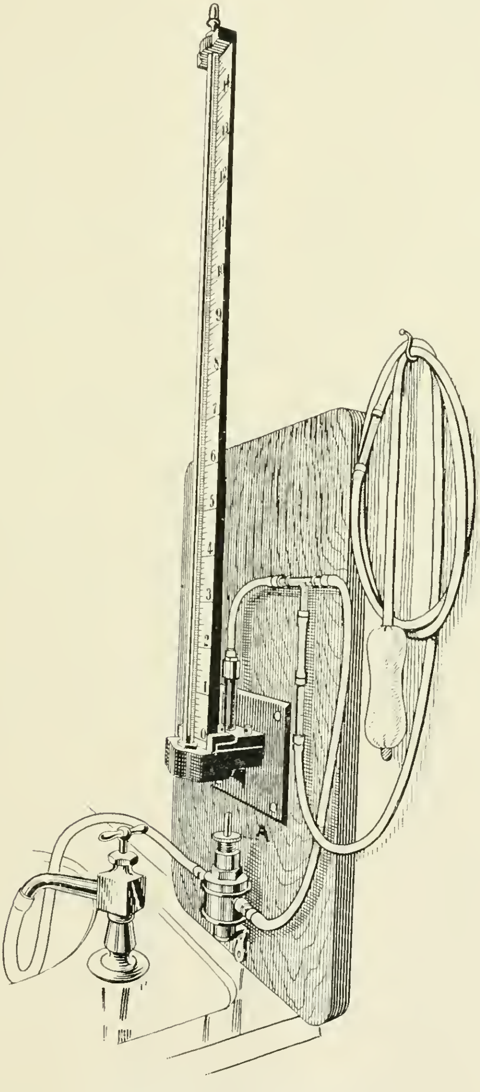
Prognosis has been considered grave in these cases, but the results gained by overstretching the cardia in cases of pure cardiospasm are more promising, although cases have, perhaps, not been observed long enough after treatment to judge if the cure will be permanent.

February 23. Dilated the cardia with rubber-silk bag dilators, using first one with a diameter of 1.5 cm., then the next size, 2.2 cm., under six pounds pressure. Patient felt the "contracting pain" when the larger size was used.

February 26. Dilated the cardia with the 2.20 cm. size, followed by the largest size, 3.00 cm., under eight pounds pressure. Patient complained of the same "contracting pain."

During the first week after the last treatment the patient regurgitated a mouthful of fluid three or four times, during the second week only once, and since then has been perfectly well and has gained twenty pounds up to the time of writing this paper.

The pronounced œsophagospasm in this case makes it rather unique. At times when I introduced the stomach tube the spasm of the œsophagus would be so severe that the patient's face became deeply congested and he could not get his breath for quite awhile, when it would come in a long-drawn whoop. When the tube had passed into the chest part of the organ it would be pushed down to the cardia with perfect ease, and the patient could drink while the tube was in place without any trouble. I frequently had to use the



Apparatus for dilating the cardia.

wire stylet to stiffen the tube to get through the upper part of the œsophagus, and at times I made several attempts before I succeeded in getting through.

In order to be able to dilate the cardia with a gradually increasing pressure, that could easily be controlled, I constructed an apparatus as follows: Between the water faucet and the quicksilver manometer, connected by rubber tubing, I inserted a pressure regulator. Between the regulator and the manometer a T-tube was inserted, the one limb of which was connected by a long rubber tube to a stomach tube having at the distal end the rubber-silk bag, as suggested by Russell[41] in 1898 (a thin rubber bag is attached to the end of a stomach tube and covered with a sausage-shaped silk bag 10 to 12 cm. long). In operating the apparatus the regulator should be set on the lowest pressure, the pressure gradually being raised by turning the screw *A* slowly from right to left. (See figure.)

To dilate the cardia forcibly in the dark is not entirely without danger, and Mikulicz and Naunyn[42] have expressed themselves to that effect, but there have been no reports of deaths from the procedure as yet. If the dilatation is gradually performed, and the pressure is under perfect control, the danger is small. A sudden high pressure would undoubtedly be dangerous, but I think this danger has been overcome by using the pressure regulator as indicated in my apparatus. The use of bougies for dilating the cardia does not give good results and are not without danger, as death from perforation with subsequent mediastinitis has been reported.

Gumprecht, cited by Strauss, says that the maximum to which the normal cardia and œsophagus can be dilated is, respectively, 3 cm. and 3.5 cm. When the expansibility of the dilator is limited and of known dimensions, the amount of pressure used is of less importance, provided the increase of pressure is gradual. When the rubber-bag dilator is not guarded the maximum amount of pressure that can be used with safety must be known. Schreiber found that the normal cardia from the stomach side could resist the pressure of 350 grams for a few seconds.

The use of the *x*-rays in making diagnosis in a number of cases has been of great help, but Rosenheim says that *x*-rays cannot always be depended upon in making a diagnosis in these cases, and that it certainly cannot take the place of the other diagnostic means in making the differential diagnosis. Krauss, cited by Richartz,[43] is of the same opinion. G. Rosenfeld, cited by Strauss,[44] used a thin rubber bag attached to a stomach-tube, which was introduced into the œsophagus and filled with the bismuth mixture in taking *x*-ray pictures. In order to determine the size of the dilatation of the œsophagus, Strauss introduced a thin rubber bag attached to a stomach tube and distended it with air. He then measured in a eudiometer the quantity of air used. Mikulicz's instrument to determine the width of the œsophagus is constructed on a plan similar to that of the Otis urethrometer. The pear-shaped rubber-silk bag sound which I used to determine the width of the œsophagus differs from Russell's dilator only in the shape of the silk bag and in that the stylet has to be in to make it stiff. The use of this instrument for sounding was suggested by Plummer,[45] who used oval silk bags.

Rosenheim and Wilms[46] have used a rubber bag attached to a stomach tube, but without silk cover to dilate the cardia (a modified Schreiber's sound), using water pressure. Strauss used air pressure to distend the rubber bag for dilatation, and he had his manometer so arranged that not more pressure than 250 cm. mercury could be brought upon the cardia. Jacobs,[47] of Antwerp, in 1898, constructed an apparatus on the plan of a urethral dilator with which he stretched the cardia to a diameter of 3.5 cm. Mikulicz has reported 4 cases in which he did a gastrotomy and stretched the cardia with a specially constructed forceps under the guidance of the fingers. Erdmann[48] reports a case treated in a similar way. Sounding without end was recommended by Lotheisen, cited by Langemak,[49] and was used by Martin in his case. In one of the cases reported by Gottstein a hard rubber tube, with a valve in the lower end to prevent regurgitation from the stomach, was left in the cardia. Jaccoud[50] mentions that Broca dilated a spasmodic stricture of the œsophagus successfully with a forceps. Cahn[51] has made use of a soft rubber tube, into which he poured mercury to dilate the cardia.

The use of the œsophagoscope will become necessary in some of the cases of cardiospasm in order to clear up the etiology, since the spasm may be secondary to ulcer or cancer of the cardia or œsophagus, and it would be rather unsafe to dilate a cardia in the presence of an ulcer.

I will include the history of another case, that is rather interesting, because there seems to be an element in the early history of the patient that points to the possibility of it being of congenital origin. As I only had the opportunity to see the patient twice, I could only make a preliminary diagnosis.

Mrs. B. S., aged 21 years, was seen January 4, 1906. Menses commenced at thirteen, always regular. One child, two years and six months old. Family history negative. She had measles, chickenpox, and whooping-cough when a child. Was never perfectly well. She had much trouble with her stomach in babyhood, and she was weak and puny. At the age of ten years she was taken out of school on account of epigastric pain and belching of gas. She never vomited or regurgitated her food at that time. From this time and until two months before her baby was born she did not have any particular trouble with her stomach. She never felt strong and well, however, and was treated for chlorosis. The present trouble began two months before the baby was born (in June, 1903). She had no morning sickness or vomiting in the first part of her pregnancy, and her appetite was good; but she commenced to experience distress, fulness, and some pain after eating, and later she began to regurgitate her food while eating. This condition has remained ever since. She says that she always regurgitates it and never vomits. The patient feels that the food does not enter the stomach. After she has regurgitated it she eats again, and the regurgitation may be repeated or the food may pass into the stomach, and better if she presses with her fingers in the epigastrium. There is a sensation of choking, oppression, or fulness in the chest on eating; she refers this sensation to a level above the diaphragm. Occasionally she is wakened in the night by slime and particles of food coming through the nose, causing a burning sensation. If she drinks water in the ordinary way it comes back immediately. She has to sip it. There is no particular food that she can take better than others. Whatever food gets in her stomach agrees with her. She never vomited blood.

January 5. I introduced the stomach tube about 12 o'clock today and drew off a considerable quantity of mucus mixed with food. In this material I found spinach that she had had for dinner the previous day. Examination of contents showed absence of hydrochloric acid and presence of lactic acid. On introducing the stomach tube it did not give the impression of entering the stomach, but it bent upon itself. Only a small quantity of air or water could be introduced before the patient gave evidence of pain.

My preliminary diagnosis was diffuse dilatation of the œsophagus or a deep-seated diverticulum. I did not have the opportunity to see the patient again. The history of this case points to dilatation of the œsophagus with cardiospasm, but the finding of spinach in the œsophagus from the previous day's meal brought the question of a low-seated diverticulum strongly into prominence. If there was not a diverticulum present, there must be either a pouch-like bulging of one part of the wall of the œsophagus or a circular gutter, as mentioned by Tyson,[52] caused by the dilated extreme lower end of the œsophagus, spreading out over the dome of the diaphragm, and in which the spinach in this case could lodge while other food had passed through. From the study of the history and limited examination of the patient, I was in favor of the latter explanation; but, diverticulum would have had to be excluded by further examination. We have here a young woman who had much trouble with her stomach when a baby and early childhood, and who had to be taken out of school at the age of ten on account of epigastric pain and belching; and then at the age of eighteen, during the latter part of her pregnancy, began to get fulness, distress, and pain after eating; and later, suddenly had an attack of cardiospasm with regurgitation of food, which condition has become chronic. It is only two years and a half since the regurgitation commenced, and yet there seems to be a considerable dilatation, which shows that there must have been considerable weakness of the wall, or that the condition is like that of Case No. 15 of Gottstein's, referred to above, in which a congenital dilatation, a catarrhal condition, had come on with subsequent cardiospasm.

I do not know of any method by which a congenital dilatation can be differentiated, except by early œsophagoscopic examination. The mere fact that the patient had much stomach trouble in babyhood and at the age of ten, does not entitle one to draw the conclusion that the case is congenital, but the history in connection with the rather pronounced dilatation which seems to be present would tend to draw the attention to congenital dilatation.

There are very few cases of dilatation of the œsophagus without stenosis reported in the American literature; besides, those above referred to I have only found the case of Liebmann,[53] in 1892, Einhorn's[54] cases in 1900, Tyson's in 1904, and Pummer's in 1906.

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PANCREATIC LITHIASIS, WITH CHRONIC INTERSTITIAL PANCREATITIS, FOLLOWED BY DIABETES MELLITUS.

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The recognition of pancreatic disease from the clinical symptoms is, on the whole, of rather rare occurrence, typical examples of this condition for the most part being found only at autopsy. We consider ourselves especially fortunate, therefore, in having been able to follow a case more or less throughout its entire course. Pancreatic calculi have been noted for many years—according to Oser—since the middle of the seventeenth century; nevertheless, the clinical recognition of the condition still remains in more or less obscurity, unless pancreatic stones are found in the stools, despite the recent efforts that have been made by Hewlett, Edsall, Schmidt, Cammage, and others, to discover definite changes in the secretions and excretions of the body. The pathological alterations of the pancreas, due to duct obstruction, have been noted by various observers, and the relationship of pancreatic disease to fat necrosis, on the one hand, and to diabetes mellitus, on the other, offers a fertile field for investigation if necessary.

The case is as follows:

H. S., married, a coachman, was admitted to the University Hospital, June 2, 1905, complaining of "pain in the upper abdomen, nausea, and vomiting." The family history was without interest. The patient had had no serious illness that he could remember. He claimed to have had a long series of malarial attacks six years previously, from which he made a perfect recovery. He had also had measles and grippe, but not typhoid fever. An accident, which befel him six years previously, is of interest, insomuch as it may possibly have some bearing on the condition; he was thrown violently from a horse, and received a severe blow in the abdomen; this was followed immediately, and for the subsequent three days, by nausea and vomiting, the vomitus containing some blood on several occasions; there was also a history of melena. These symptoms subsided in a week or ten days, and did not return.

The dyspepsia and abdominal pain, for which the patient applied for relief, had come on insidiously, about eighteen months previously, and they were described as paroxysmal rather than as continuous, each attack being rather more severe than the one before. The present one dates back about two weeks, and was chiefly characterized by abdominal pain in the upper quadrants, associated with a feeling of oppression about an hour after eating. These symptoms were never considered alarming by the patient, nor did they give him any marked discomfort, and were generally relieved by some simple remedy. For the past week they have become more severe, the nausea and vomiting most distressing, and the bowels obstinately constipated.

The man was well developed; there was no evidence of loss of flesh, the muscles were hard and firm; the skin elastic; no jaundice; the lips and mucous membranes were of good color. The heart and lungs were normal. The abdomen was soft and natural looking, nowhere tender except along the right costal margin and epigastric regions; the patient invariably winced when pressure was made in these regions. The temperature was normal during the stay in the hospital. Urinalysis showed: specific gravity, 1024; reaction, acid; no albumin or sugar; microscopically, negative. Subsequent urinary examination was invariably negative during this first stay in the hospital. The stomach contents after a Boas test-meal showed the following: total amount withdrawn, 125 c.c.; total acidity, 32 c.c.; free hydrochloric acid, 10 c.c.; no lactic acid. Microscopically: mucus, starch granules, fat droplets, and muscle fibers.

The case was looked upon as one of indigestion with subacidity, or possibly gallstones in the gall-bladder or the cystic duct. Treatment was at once begun, the pain and nausea soon subsided, and the patient was up and about the wards in a few days feeling much benefited. On June 14, after having experienced some pain during the night, he was put to bed for examination, and the following note was made: "The patient has been doing well for the past week. This morning he complains of a continuous pain in the epigastric region, and along the right costal margin. On examination, there is to be felt in the lower epigastric region, on deep palpation, a firm, resistant mass, which can be rolled under the finger. Pressure on this mass causes the patient a distinct amount of pain; it is elongated, extending from side to side, and is apparently about the size of a fountain pen. The percussion note over it is rather dull."

The urinary examination proved negative at this time as did the stools.

Subsequent examination, when the stomach was empty, always revealed the mass. It disappeared, however, when the stomach contained food. The patient was discharged on June 22, having been in the hospital twenty days.

He was re-admitted on January 31, 1906, after an absence of seven months and eight days, complaining of "general weakness, frequency of micturition, cough, and loss in weight." He said that the great loss in his strength, and polyuria, had come on rather suddenly about four months previously—that is, some time in October, 1905, and that since that time the loss had been steady. A cough, which had developed a month previously, had markedly increased in frequency and violence; the expectoration, watery and thin at first, had gradually become thick and purulent. His weakness, which was steadily progressive, often compelled him to remain in bed several days at a time. Urination was very frequent, often seven or eight times during the night.

The patient's statement that he had lost 42 pounds in weight could readily be believed; there was marked evidence of loss of flesh; the lips and mucous membranes were pale, and the eyes sunken. At the apex of the right lung tubular breathing was heard as far down as the third rib, and the breath sounds were accompanied by showers of fine crackling rales; percussion over this area was distinctly dull. Hepatic flatness began at the lower margin of the seventh rib and extended to within 2 cm. of the costal margin (for the most part, the liver dulness was almost obliterated by the abdominal tympany). The abdomen was markedly distended, especially in the upper quadrants; this distention was remarkable, and was a constant feature up to the day of the patient's death, and caused marked embarrassment to respiration. The tympany over the abdomen was drum-like even in the flanks, and the superficial veins were tortuous and prominent in the upper abdomen, especially along the costal margins. On palpation, nothing of importance was to be made out, the mass which had been so easily felt during the patient's first stay in the hospital was not palpable; there were no areas of tenderness, except in the epigastrium and along the right costal margin, when the patient would wince during examination, but more, apparently, from the discomfort occasioned by pressing on a distended gut than from actual pain.

There was no ascites, and no fluctuation could be elicited anywhere. The genitals were normal, and there was no œdema nor glandular enlargement. No jaundice was ever noted (nor could any such history be obtained). The urine was clear and limpid, specific gravity 1028, acid in reaction; microscopically it contained epithelial cells, mucus, cylindroids, and an occasional pus corpuscle. There was a faint trace of albumin and a decided reaction for sugar by Fehling's test. A leukocyte count showed 15,000 per millimeter. The sputum was mucopurulent, and contained many tubercle bacilli. From January 31 to February 16 (the day of death) estimations of the total amount of urine, and the gross and microscopic appearances of the stools were carefully noted; both presented a most unusual picture, especially the stools. The total daily urinary output averaged about 4000 c.c., the maximum being in the neighborhood of 6000 c.c.; while the estimations of sugar, though varying in amounts, according to the diet, was always high. On a diet consisting of bread, meat, potatoes, etc., given ad libitum, the total sugar output was found to be around 7 or 8 per cent. (by the polariscope); a diet as free of carbohydrates as possible, though reducing the sugar to 2 or 3 per cent., would never altogether clear it. Acetone and diacetic acid were never present, even toward the terminal stages of the disease.

The stools were, perhaps, the most remarkable feature of the condition. They were, for the most part, typical examples of pancreatic disease; they varied in amount, according to the diet and general condition of the patient. Under a full and unrestricted diet they were enormous, the total amount of fecal matter collected under these conditions in twenty-four hours was 4 liters on one occasion. The daily output, however, varied, as might be expected, according to the amount and character of food given. In appearance, they were soft or semisolid, of a leaden-brown color, while here and there scattered promiscuously about, were lumps of cheesy-looking material, yellowish-white in color, and varying in size between an English walnut and a split pea; these masses proved upon microscopic examination to be lumps of fat. Another remarkable feature was the collection of an oily viscid material, dark yellow in color, around the margin of the vessel which contained the stool. The odor was very penetrating, and was in no sense of a fecal character; it was of a distinctly rancid butter odor, and could be detected in the furthest corner of the laboratory. Microscopically, a fresh stool showed large quantities of free fat, a great amount of unaltered muscle fiber, and debris.

A chemical analysis made by Dr. C. P. Emerson showed the following: Of the total stool (dried)[1] fat, 54.6 per cent.; neutral fat, 22.5 per cent.; fatty acids, 32.1 per cent. In 5 grams (weighed after drying the evaporated stool over sulphuric acid) there was: neutral fat, 1.128 grams; split fat (fatty acids), 1.6376 grams. Microscopically, there was fat as globules and as crystals.

The urine showed Hewlett's reaction for lipase on a number of occasions in specimens rendered free of sugar; negative results were invariably obtained, however, when the reaction was looked for in sugary urine.

Our results with Schmidt's meat-test were positive. This test, it must be said, was only tried during the last two weeks of the patient's life, and when the diagnosis of pancreatic changes were certain, and when, as was afterward learned by the autopsy findings, pancreatic juice must have been entirely absent from the alimentary tract, on account of complete occlusion of the duct of Wirsung by a stone. The meat-balls, sewn in silken bags, were readily recovered in the stools between six and twelve hours after their administration. The outside layer of the meat-ball showed complete absence of the nuclei of meat fiber, while the inside, protected as it was from bacterial processes, showed the nuclei universally intact.

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[1] According to Emerson, Müller showed that while 84 per cent. of the fat was normally split, if the pancreatic juice be excluded, there is only about 40 per cent. This low percentage of split fat in the stool forms, according to Kinnicutt's statement, the only sure sign of absence of pancreatic juice.

The temperature from the time of entrance of the patient to his death was elevated, running a more or less typical hectic chart. The pulse varied between 70 and 120. A summary of the clinical findings is as follows: A man approaching middle life, a well-defined history of a gradually increasing dyspepsia, with a history of a violent blow in the abdomen; the finding (seven months previous to first admission to the hospital) of a well-defined mass in the upper abdomen, corresponding in main to the clinical position of the pancreas; the sudden onset of, and gradually increasing polyuria (a symptom from which we have the right to draw the conclusion that the diabetes was more or less of sudden onset); marked loss of flesh, and a corresponding loss of strength; the development of pulmonary tuberculosis, with rapid cavity formation (about four months, according to the history), in all probability the formation of the lesion being hastened by the enormous quantities of sugar; ballooning out of the intestines; large semisolid stools, containing relatively large quantities of unaltered muscle fiber, together with superabundance of free fat, and low percentage of split fat (32.1 per cent); the presence of Hewlett's lipase reaction; and the positive results obtained from the meat-bag test of Schmidt, made a clinical picture not often seen, and one from which we drew the conclusion that we were dealing with occlusion of the duct of Wirsung, and that operative procedures might be of benefit. Unfortunately, however, the large quantities of sugar in the urine (regardless of diet), and the ever-increasing pulmonary symptoms, made the operative chances practically nothing. The patient died suddenly while walking about the ward.

Autopsy. Body emaciated. Small amount of subcutaneous fat. Heart and pericardium normal. Lungs: In the upper lobe of the right lung a large tuberculous cavity is present. The entire lung is bound down by dense pleural adhesions. The liver and kidney show some evidence of parenchymatous degeneration. The stomach and intestines are normal. The pancreas is very small and hard. Rather firmly adherent to adjacent parts. It is dense and nodular, and has a stony hardness. It is 13 cm. long and 2 cm. wide, and weighs 35 grams. The duct of Wirsung is very much dilated, and will readily admit a large lead pencil (Fig. 1). About 2 cm. from the termination of the duct there is lodged a calculus, roughly about the size of an orange seed, very irregular in outline, presenting numerous small spicules, which completely occlude the duct. When the duct is opened, about 3 c.c. of turbid fluid, which was dammed back, flows out. This fluid contains a quantity of gritty particles (sand), which are scattered throughout the duct and its tributaries. The duct of Santorini is not to

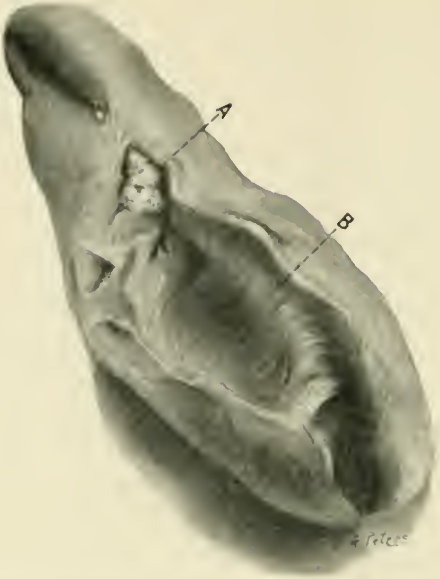

Fig. 1.—Pancreatic lithiasis, showing marked dilatation of the duct of Wirsung. *A*, stone; *B*, dilated duct.

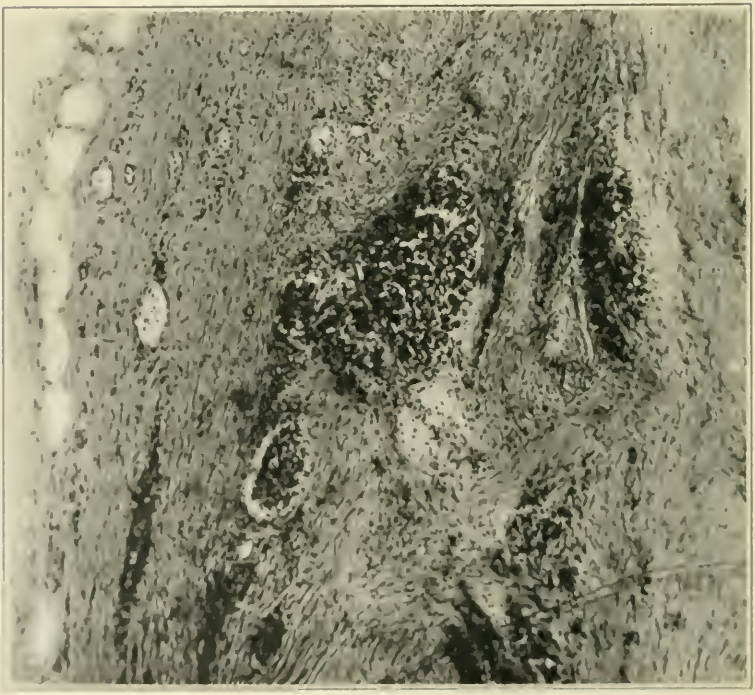

Fig. 2.—Marked fibrosis of the pancreas.

be made out, being obliterated and enveloped in the dense connective tissue which is scattered throughout the organ and in large part replaces the gland tissue.

Anatomical Diagnosis. Tuberculosis of the lung; sclerosis of the pancreas; pancreatic calculus; parenchymatous degeneration of the liver and kidney. The microscopic examination of the organs, except the pancreas, showed nothing worthy of note.

Microscopic Examination of the Pancreas. The pancreas is almost wholly transformed into connective tissue, and presents the appearance resulting from duct obstruction (Fig. 2). The parenchyma is in great part destroyed, so that in places only islands of Langerhans persist; this point is especially well shown in the specimens. However, in places the islands seem to have been destroyed along with the parenchyma; embedded in the scar-like connective tissue, they are compressed and the nuclei are small and in places are invaded by connective tissue. Some of the islands are hypertrophied and their form altered. Areas of hyaline degeneration are not to be made out. Here and there are seen one or two small ducts, either in transverse or longitudinal section. In certain fields one sees a small patch of pancreatic tissue, the cells of the acini showing evidence of degeneration. The condition is evidently one of advanced sclerosis of the pancreas, and, indeed, the lesion is so far advanced that it is impossible to recognize it as a definite type. The connective tissue not only extends between the lobules, but also invades the acini, dividing the parenchyma into small groups of cells, such as are seen in advanced cirrhosis of the liver, to which this condition seems to be in every way analogous.

Despite numerous experiments, autopsy studies, and clinical observations, the relationship between pancreatic disease and diabetes mellitus cannot be said to be definitely established, either as to its anatomical or physiological changes. Scientific knowledge dates from the experiments of Minkowski and von Mering, who succeeded in producing permanent diabetes in dogs by total extirpation of the pancreas; they also showed if a small portion be left behind at the time of the operation, diabetes will not occur, even though this portion be separated from the duodenum and the pancreatic duct be ligated. They furthermore showed that if after extirpation of the pancreas a small piece of pancreatic tissue be inserted under the skin of the abdomen, diabetes would not occur. They, therefore, concluded that a "something" is formed in the pancreas which passes into the circulation, and brings about sugar destruction, or prevents the accumulation of sugar in the blood.

Langerhans, in 1869, described groups of cells, irregularly polygonal and having a rounded nucleus situated in the parenchyma of the lobules. These structures are termed the islands of Langerhans. Probably they have the same origin as the acini, but, according to Opie's observations, they do not communicate with the ducts of the glands, and are independent of them; they are surrounded by a capillary network, so that their secretions may readily be taken up by the blood. Herxheimer claims to have been able by serial sections to trace definitely the transition from the islands of Langerhans to the acini, and, in contrast to Opie, claims that they are not isolated structures. These islands are three times more numerous in the tail than in the head of the pancreas, and doubtless have some relationship imperfectly understood to carbohydrate metabolism. Lewaschew claimed he could increase their number by overstimulating the gland with repeated injections of pilocarpine, and that this increase took place at the expense of the acini; but Opie, by a series of experiments, found no such alteration taking place.

As to the function of these structures, it seems to be the accepted opinion that they are in some way associated with carbohydrate metabolism, and that diabetes is either dependent on some interference with the secretion of the pancreas, due to degeneration of these islands, or to the production of an excessive internal secretion by the islands—or, as Herxheimer states it—diabetes is due not so much to an anatomical change in the pancreas as to an alteration of the functional activity of the pancreas remaining.

The etiology of chronic interstitial pancreatitis is often obscure. It is probably a more common disease than the number of reported cases would seem to show, and many of the cases diagnosticated as cancer of the head of the pancreas are, doubtless, of this character. In 3 cases of chronic interstitial pancreatitis detected by the stony, indurated feel of the pancreas during operation for gallstones, Riedel feared malignancy, but 2 of the patients recovered and remained well for a number of years, thus disproving the idea of malignancy, and the diagnosis of the third case was confirmed at autopsy. The disease is one of the middle period of life, two-thirds of the cases occurring between forty and sixty years. One of the commonest causes is duct obstruction, due either to a pancreatic calculus in the duct of Wirsung, as in the case under consideration, to gallstones in the common duct or in the ampulla of Vater, or to neoplasms, usually malignant, obstructing the outflow of pancreatic juice. Bacteria reaching the gland, either through the duct or through the blood stream, may give rise to a chronic hyperplastic inflammation, as in the case reported by Moynihan, in which the condition followed typhoid fever and was due to the typhoid bacillus. Syphilis, tuberculosis, and alcohol may occasionally produce the condition.

As to the histological changes seen in the pancreas in diabetes, the work of Opie has cast much light. He distinguishes two forms of chronic pancreatitis: (1) Interlobular pancreatitis, characterized by proliferation of fibrous tissue between the lobules, which are invaded from the periphery, and (2) interacinar pancreatitis, in which the newly-formed fibrous tissue is more diffusely distributed within the lobules and between the individual acini. In the first type, which follows occlusion of the pancreatic duct, the islands of Langerhans are implicated only when the sclerotic process has reached an advanced grade. In Opie's studies he noted but 1 case of duct obstruction due to calculus, with an accompanying diabetes. It was very similar to the case we report, differing, however, in the fact that the glycosuria was of mild character, the sugar disappearing when carbohydrates were withdrawn from the diet. On the other hand, in 2 of his cases with advanced sclerosis following obstruction of the pancreatic duct (1 case caused by calculi, the other by carcinoma) diabetes was absent. Hyaline transformation of the islands without any definite change in the parenchyma has also been noted by Opie.

On the other hand, several observers, notably Hanseman and Herxheimer, contend that the changes in the islands of Langerhans are of but little importance in the production of diabetes, and contend that the alteration in the parenchyma is of prime importance in the production of sugar in the urine.

As far as a study of one case permits, we are inclined to the view that sugar does not appear in the urine until the islands of Langerhans become involved in any disease of the pancreas. The clinical history of this case shows a man who has suffered from disease of the pancreas for some time. At the time of entrance into the hospital he showed evidence of this disease by the characteristic condition of his stools and the presence of a hard, nodular mass in his abdomen, which proved to be pancreas. The organ at this time was evidently markedly sclerosed and the parenchyma involved. It has been shown that the islands of Langerhans resist sclerosis following damming back of secretion upon the gland and finally suffer only when the acini are almost entirely destroyed and replaced by dense, scar-like tissue. However, at this time no sugar was present in the urine, although numerous examinations were made extending over a period of thirty days, with the patient on a full hospital diet. Seven months later, however, sugar was found in the urine and the symptoms of diabetes seemed to appear rather suddenly. From a study of the microscopic specimens, we believe that at this time the islands began to be involved in the sclerosis, and, therefore, the normal carbohydrate metabolism was disturbed.

TYPHOID FEVER IN THE AGED.

By Arthur S. Hamilton, M.D.,

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In the *Boston Medical and Surgical Journal* for 1842 Elisha Bartlett reported an attack of typhoid fever occurring in a woman aged sixty-three years. In commenting on this case he said: "The principal interest of this case is connected with the age of the patient. It is now well settled that typhoid fever is, in the great majority of instances, a disease of early life. Of 138 cases, the histories of which are analyzed in Louis' *Researches*, only 10 were over thirty years old and no one was over forty. Of 147 cases cited by Chomel only 6 were over forty years old and only 1 was over fifty. The last-mentioned writer says, in his *Clinical Lectures on Typhoid Fever*, published in 1834, that so far as was then known no case had been met with in which the patient was over fifty-five years of age. In what Osler[1] describes as the first articles ever published which clearly set forth the difference between typhoid and typhus fevers, Gerhard, of Philadelphia, had said that the age of those affected with typhoid rarely exceeded thirty years. With these opinions Bartlett did not wholly concur, and, in the course of his article, he stated that he believed this disorder to be of more frequent occurrence in those of advanced years than was generally held to be the case. He thought that the susceptibility to the disease, in an unprotected person, did not diminish with increasing age and believed that its comparative infrequency in advanced years was due to the fact that so many had suffered from it in an earlier period of life, it being then thought that no one could contract typhoid fever the second time. This opinion of Bartlett, however, would seem not to have been generally accepted, for, several years later, George B. Wood,[2] in speaking of typhoid fever, said: "It is comparatively rare beyond thirty years, much more so beyond forty, and very rare indeed beyond fifty." Since Wood's time, however, a number of articles have appeared calling attention to the relation between old age and typhoid fever, and, even previous to the appearance of Bartlett's article, Prus[3] had reported an attack in a woman of seventy-eight. In 1859 Uhle[4] contributed a lengthy article on typhoid fever in old people, and in 1888 Lautre[5] published a similar article. In February, 1898, Morris Manges[6] called attention to the matter and reported 5 cases in individuals past sixty, all coming under his observation during the year 1897. He thinks that the disease is not infrequent in the aged, and that its presence is often overlooked. Most authorities, however, still consider typhoid as occurring very rarely in old people, and the general opinion is that it is rarely seen. The number of isolated cases on record, depending for their interest on the age of the patient, would seem to indicate this. Osler[7] states that of 829 cases admitted into the Johns Hopkins Hospital up to May 15, 1899, there were 6 between fifty and sixty and 6 above sixty. He agrees with Manges that, as the course of the disease is often atypical, the diagnosis will be uncertain, and, in this way, cases will be overlooked In 2 of his cases the disease was not recognized until the autopsy was made. In the article mentioned above, Manges quotes from the statistics of the New York Board of Health, and says that out of a total of 3634 deaths from typhoid fever, occurring in that city from 1887 to 1896 inclusive, 96, or 2.6 per cent., were in persons over sixty-five, and 414, or 11.3 per cent., were in persons between forty-five and sixty-five. Even allowing for the high death rate in the aged, this evidently means a considerably higher percentage of typhoid fever in old people than is ordinarily derived from general hospital statistics.

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[1] A Text-book of the Practice of Medicine, p. 2.

[2] Practice of Medicine, 5th ed., i, 351.

[3] Rev. méd. franç. et étrang., Paris, 1837, i, 62.

[4] Arch. f. physiol. Heilk., Stuttg., 1859, N. F., iii, 76.

[5] Rev. méd. de Toulouse, 1888, xxii, 245.

[6] Med. Record, February 26, 1898.

[7] Johns Hopkins Hosp. Reports, viii, 421.

In continuation of the work of Manges, I have collected statistics of the Health Departments of Chicago, Boston, and St. Louis, kindly placed at my disposal by the city physicians. The results are similar to those reported by Manges.

Table I.—*Department of Health, Chicago.*

Year.	Deaths all ages.	Over fifty years of age.
1897	437	56
1898	636	47
1899	442	57
1900	337	37
1901	509	48

Table II.—*Department of Health, Boston.*

Year.	Deaths all ages.	Over fifty years of age.
1897	173	34
1898	185	20
1899	165	12
1900	143	8
1901	142	9

Table III.—*Department of Health, St. Louis.*

Year.	Deaths all ages.	Over fifty years of age.
1897	124	6
1898	95	6
1899	131	3
1900	168	16
1901	198	10

Total number of deaths, all ages, 3885. Total number over fifty years, 369. Percentage of cases over fifty years, 9.5.

In an epidemic of typhoid fever which occurred at the Hospital for the Insane at Independence, Iowa, a number of people quite well advanced in years contracted the disease, and a study of the statistics of these cases leads me to believe with Manges and others that those in middle and advanced life are much more likely to be attacked than is usually conceded to be the case. Out of a total of 188 cases of typhoid fever studied at the Independence Hospital, 27, or 14.3 per cent., were in individuals fifty years of age or over.

TABLE IV.

Ages.	Cases.	Actual ages.
50 to 54 .	6	50, 51, 51, 52, 53, 53.
55 to 59 .	8	56, 56, 57, 57, 58, 58, 59, 59.
60 to 64 .	9	60, 60, 60, 61, 61, 61, 61, 63, 63.
65 to 69 .	1	65.
70 to 74 .	2	71, 71.
75 to 79 .	1	78.

The mere fact that in so large a percentage of these cases the individuals were well advanced in years, however, cannot be taken as of much value in determining the liability of aged people to contract typhoid fever, since there were very few in the hospital below twenty years of age, and the presence of young people in the proportions in which they are found in general medical work would undoubtedly lessen very considerably the relative number of old people who became ill, so that these statistics cannot with justice be compared with those of the community at large. I have, however, studied more carefully the individuals in certain parts of the hospital. For this purpose, I took those who were living in two cottages and in two wards, in all of which typhoid fever was particularly prevalent. The patients situated in this way were all on the male side of the hospital. In the above-mentioned parts of the institution there were, in all, 318 men, of whom 65, or 20.44 per cent., contracted typhoid fever. So far as known, there was nothing in the surroundings or condition of these 65 patients which would render them any more susceptible than the others. Below is a table showing the number and percentage of those who, at these ages, developed typhoid fever.

TABLE V.

Ages.	Cases.	Those who developed typhoid.
20 to 29 .	55	15 or 27.27 per cent.
30 to 39 .	89	24 or 26.96 "
40 to 49 .	78	17 or 21.79 "
50 to 59 .	44	5 or 11.36 "
60 to 69	11	2 or 4.87 "
70 to 79	8	2 or 25.00 "
80 to 89	3	0 or 0.00 "

Average age of all, 43.13 years.

Average age of those who contracted typhoid, 38.9 years.

The small number of cases considered makes it impossible to draw any very positive conclusions from these statistics. Yet, in so far as they can be relied upon, they would seem to show that the liability to typhoid fever is very little less from thirty to forty than it is from twenty to thirty, and that the liability from forty to sixty is altogether beyond what one would gather from most statistics.

While assuming, therefore, that there may be considerable error involved in these figures, I am still of the opinion that the immunity to typhoid fever conferred by advancing age is less than is generally supposed, and I believe that statistics gathered from patients in private life would produce somewhat different results from those which we now have, which are almost invariably taken from general hospitals, where, as Curschman[8] states and shows by statistics, the class of patients received does not represent the average population of a community, since young or middle-aged unmarried people are much more likely to be admitted than older people.

In considering the clinical course of these cases, my attention has been drawn to a number of points in which the symptomatology has differed from that of the classical form of typhoid fever as observed in younger people. Not all of the 27 cases ran this atypical course, yet marked variations were common. In 3 cases, at least, the degree of aberration was so great that they would not have been included in the list of typhoid patients had not the subsequent development of a Widal reaction, not present at the outset, made positive the diagnosis. All the cases, however, differed widely from one another, so that it would be very difficult, indeed, to construct a type case of typhoid fever for an old person. In a general way, it may be said that the signs in the aged differ from those of younger people in that those on which we ordinarily depend largely for diagnosis, namely, a certain type of fever, rose spots, enlargement of the spleen, and nose-bleed, are frequently absent; whereas, the various complications which, at times, are so misleading in diagnosis, are likely to be present to a marked degree.[9]

Inasmuch as the patients whom I had to study were chiefly cases of senile dementia, or of terminal dementia of a high grade, it was difficult to elicit fully the subjective signs of the onset of the attack. Yet from the information at my command, I am inclined to believe that the prodromal period is lengthened, though the headache, nose-bleed, general malaise, chilly sensations, and pains in the back and legs, so prominent in younger people, are usually present in mild form or else are largely absent; thus, nose-bleed during the prodromal period, was not noticed in any case. In two instances, altogether unusual premonitory symptoms were present. In a man of fifty-eight, not an epileptic, the first thing noticed was an epileptiform convulsion. No diagnosis was made in his case until a Widal reaction was sought for and obtained, one week later. A woman of fifty-nine presented at the outset typical signs of uremia, except that she had a temperature of 103°, and the urine gave a diazo reaction. Several days later a Widal reaction was obtained in her case also.

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[8] Nothnagel's Encyclopedia, Typhoid and Typhus Fevers, pp. 56, 59.

[9] It is proper to state that Curschman considers complications in this class of cases relatively infrequent, and Lautre says that they generally do not occur; but this is quite contrary to the ideas of others who have contributed to the subject and to my own experience.

During the attack that followed there was but little in her symptoms that pointed to typhoid fever, though two well-marked subsequent relapses were quite typical; throughout her illness, however, the signs of renal involvement continued very prominent.

When the disease had become fully established, the temperature curves rarely conformed to the classical type. In only 1 of the 27 cases was there any marked tendency to the production of this well-known curve. The ordinary diurnal variation is, in old people, much increased, and in almost every one of the 27 cases the temperature was distinctly remittent, even from the onset conforming closely to Strümpell's period of steep curves. In 7 cases it was even intermittent, reaching the normal two or three times during the first week.

In 5 cases the highest temperature recorded was between 105° and 105.8°. In 15 cases it was between 104° and 105°; in 5 cases, between 103° and 104°; and in 1 it was 102.6°. The average temperature in these cases, however, was by no means so high as this. As a usual thing it did not go above 102° or 103°, and in a few cases it was even lower. Along with a low grade of febrile movement it was noticed that the period of lysis came unusually early. Defervescence usually began so soon that by the eighth to the eleventh day the temperature would be normal or practically so, yet it would rarely remain there, and a marked tendency to great prolongation of slight febrile movement was observed. In only 3 cases did the temperature remain substantially normal after the first reaching that point. In 7 cases after reaching normal, there were rather slight variations extending over a considerable period, and in 11 cases there were one or more distinct recrudescences in addition to these slight variations. Of the latter, 1 case underwent also two distinct relapses, and 2 cases had one relapse each; the remaining 6 terminated fatally. Curschman refers to this irregularity of the temperature, and says:[10] "Not a few cases pursue so anomalous a course that the form of the curve furnishes no diagnostic information whatever, but rather may lead to incorrect conclusions." He also states, however, that the frequency of relapses and recrudescences diminishes distinctly with increasing years—a finding quite at variance with mine. Osler [11] reports a case with several normal or subnormal temperature records during the early part of the disease, and almost every writer on the subject refers to the striking irregularity of the temperature.

Chills and sweats seemed also to be more common in the aged than in younger individuals. In 6 of my cases there were well-marked rigors during the course of the disorder, all accompanied by profuse perspiration, and in 1 other case there was profuse perspiration without rigor. These were not mere chilly sensations, and were not produced by the baths. In a case complicated by menin-

[10] Nothnagel's Encyclopedia, Typhoid and Typhus Fevers, p. 301.

[11] Johns Hopkins Hosp. Reports, iv, 29.

gitis, to be described later, chills and sweats were very pronounced. At the same time there was very great irregularity in the fever, the rectal temperature at one time during the height of the disorder being only 95.6°. On the evening of the same day it was 104.6°, a rise of 9° in twelve hours.

Functional disturbances of the circulation were also very common, and in 70 per cent. of the cases the pulse was described as bad at some time during the disorder. In 39 per cent. of the cases there was distinct intermission of the pulse. Even in those individuals in whom the general manifestations of the disorder were quite mild the pulse, though strong and regular at the outset, would fall after a couple of weeks out of all proportion to the severity of the general symptoms, and it was also noticeable in a number of individuals that the pulse did not return to the normal for some time after the fever had subsided and the patient seemed to be doing nicely in other respects. Two patients had a mitral systolic murmur and one had an aortic systolic murmur. These were, doubtless, all organic in origin. A fourth patient had an aneurysm of the right common carotid. In 1 individual of fifty-nine there was a very high grade of arteriosclerosis, the radial arteries being typically beaded. The pulse in this patient was very intermittent throughout the attack and, on several occasions, almost imperceptible. In spite of two well-marked syncopal attacks, the patient recovered, and is now in fair health. Another man had phlebitis of the left saphenous and left external and common iliac veins, and died on the twenty-seventh day. A marked slowing of the pulse during convalescence is reported as common by some writers, but it was observed in only 2 of my cases, and even then the pulse did not go below 50.

The Widal reaction was present in every case, but as the examination was made merely for its scientific interest in most instances, and as the tests were carried out only as time permitted, I can say nothing as to the period of the disease at which the reaction first appeared.

Disturbances of the respiratory system, in my cases, were quite as common as those of the circulation, though Lautre says that usually only bronchitis or slight congestion is observed. Cough was present in about 90 per cent. of the cases, and rales were observed a number of times, but these were not studied with sufficient care to enable me to gather percentages. Pneumonia was present as a complication four times, once at the outset of the fever and three times during convalescence. In 1 of these cases pleurisy was severe, and in another case pleurisy was present without signs of pneumonic invasion. In two individuals who died, small tuberculous foci were found postmortem, but these doubtless exercised no influence on the course of the disease. Epistaxis was not common, being present in only 12 per cent. of the cases. The infrequency of epistaxis in old people has been noticed by other observers.

Marked disturbances of the alimentary tract, though less common than those of the lungs, were still frequent and severe. The appearance of the tongue did not differ from that ordinarily seen, except that it seemed a little more tremulous, more deeply furrowed, and more often dry. Eleven out of 27 vomited more or less at some time during their illness, and some to a very marked degree. Two, on account of their mental condition, refused absolutely to take any food or medicine, and had to be fed by the tube. After the third feeding one of these began vomiting, and continued so throughout, vomiting everything put into his stomach. He was next fed by the rectum for a couple of days, but his rectum soon became so irritable that it also rejected everything put into it, and in a short time the man died from combined inanition and organic heart-failure. This man was seventy-eight years of age, the oldest of the series. The other man who had to be fed with a tube began to take milk by mouth after a time, but eventually developed pneumonia and died on the thirty-first day. During the last five days he had severe hiccoughs, which continued, at times, for as long as six hours without interruption. He was seventy years of age.

During the prodromal period of the disorder constipation was more frequent than diarrhœa, 71.4 per cent. being classed as constipated; but, as soon as the disease was well established, diarrhœa became very common. Dreschfeld[12] says diarrhœa is frequent, but not usually profuse. Wilson,[13] Hutchinson,[14] and Murchison[15] think it less common than in younger people. Josias[16] and Lautre say it is always present and Curschman[17] thinks there is no difference between old and young in this respect. In 11 of my cases diarrhœa was present throughout the attack. In 7 it was present nearly all the time. In 3 cases there was persistent constipation, in 3 others the patient alternated between constipation and diarrhœa, and of 3 there is no record. The stools in the cases of diarrhœa were often of a peculiarly fœtid character.

In 18 cases tympanitis was quite marked, and in one instance the distention was very great. The unusual number of individuals who suffered from abdominal distention is probably accounted for by the general flabby condition of the tissue of old people, as well as by the intestinal disturbance. Hemorrhage from the bowels occurred three times, in 1 patient once, in another patient three times, and in a third patient four times, in the last instance resulting in death. Perforation, with subsequent peritonitis, was present but once. This case also terminated fatally. Enlargement of the spleen is said by some authors to be quite uncommon among old people affected with typhoid fever, yet in our cases it was made out in about 40 per cent., and the postmortem examinations in the 6 cases which came to autopsy bore out this finding. In 2 of the 6 cases the spleen was very small, in 2 it was slightly enlarged, and once it was very large, weighing 715 grams. In the 27 cases reported herewith, jaundice was present three times. All 3 patients showed symptoms of cholecystitis, from which all recovered without operation. As 3 other cases of jaundice occurred during this epidemic, all in patients a little below fifty, and, as Osler says, that out of the first 500 cases of typhoid admitted to Johns Hopkins Hospital there was no case of jaundice, the presumption seems a fair one that infections of the biliary apparatus are more common in the aged than in the young, though a study of Camac's[18] and Keen's[19] statistics fails to bear this out.

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[12] Allbutt's System of Med., p. 834.

[13] Amer. System of Practical Med., i. 203.

[14] Pepper's System of Med., i. 242.

[15] Quoted from Lautre, Ibid.

[16] Thèse de Paris, 1881.

[17] Nothnagel's Encyc., p. 342.

As might be expected, disturbances of the genito-urinary system were also frequent. The diazo reaction was obtained 16 times and 6 times it was absent. In the 6 cases repeated examinations were made, so that it can be definitely stated that no reaction was present. In 5 cases there is no record of any adequate examination. In 22 cases in which the test for albumin was made it was found present every time, 13 times in considerable or large quantities and 3 times in small amount. In 5 cases there is no record of any adequate examination. Casts were found 14 times, and were positively excluded 8 times. In one instance hyaline casts only were found; in all the others pale and dark granular casts also were observed. In a considerable number of cases there was retention of urine, requiring catheterization, and, probably partly in consequence of this, pus in considerable amount was found in the urine of a good many patients, though this was present several times when no catheter had been used. Curschman[20] thinks that febrile albuminuria occurs with the same frequency as at the other periods of life, and that typhoid nephritis is met with much less often in old people.

In the seventy-eight-year-old patient who died from inanition and heart trouble, bilateral orchitis developed early in the disease. It had begun to improve somewhat at the time of his death. In a case mentioned at the beginning of the paper, the symptoms of renal disturbance were marked throughout. When first noticed, the patient was very pale and showed moderate œdema, which rapidly increased. There was total suppression of urine for a time, and what was passed subsequently contained a considerable number of casts and a moderate amount of albumin. The œdema persisted throughout the attack, which lasted, with two relapses, 124 days. A peculiar feature in this case was that the diazo reaction persisted after the temperature had fallen below 101°, something which I have not seen present in any other case.

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[18] Johns Hopkins Hosp. Reports, viii, 339.

[19] Surgical Complications and Sequelæ of Typhoid Fever, p. 263.

[20] Nothnagel's Encyclopedia, Typhoid and Typhus Fever, p. 343.

Notwithstanding the already disturbed mental state of these patients, symptoms connected with the nervous system were not common. In 3 there was mild delirium and 3 were quite stuporous. Several were quite restless and were with much difficulty kept in bed, but this was probably not much more noticeable than would have been the case had the same individuals been confined to bed from any other cause. In a few cases there was a very striking mental improvement following the onset of the fever. General tremors were very common, as might be expected. As already mentioned, in 1 instance the illness was first noticed after an epileptiform convulsion. This man was probably well along in the first week of the attack. One patient, with true typhoid meningitis, presented marked mental symptoms. Throughout her attack she was very restless and emotional, and there was a pronounced general tremor. Her pupils were very sluggish and at the last were firmly contracted. Cultures made immediately after death from the brain cortex of this woman revealed pure typhoid bacilli, and, later, bacilli resembling typhoid germs were found in the brain tissue itself.

It seems to be quite generally accepted that rose spots are less common in old people than in the young, and in this series they were present only five times, twice in large numbers, and three times in small numbers. One patient presented quite large patches of erythema, which were present only two days.

Convalescence was an exceedingly tedious process, for, as already mentioned, when the temperature had about reached normal it would still persist in slight elevations, sometimes for many days. In those cases especially in which the attack had been severe the patient was left very weak and considerably emaciated. In such individuals, when lying in bed, disturbances of the lungs, which delayed the period of convalescence, developed several times. One patient who had seemed to be convalescing fairly well, though still very weak and lying helplessly in bed, suddenly developed pneumonia and died. Two cases were much delayed in convalescence by attacks of cholecystitis. Setting the limit of recovery at the time when the patient was able to be dressed and walk about, the longest illness was 124 days. This was in the woman who presented symptoms of uremia and who had two relapses. The next longest illness was 120 days, and was in one of the women who developed cholecystitis during her convalescence. The shortest illness was 26 days, and the average about 60 days. It has been said by some that marked subnormal temperatures are characteristic of the period of convalescence, but this has not been my experience. In 3 cases the temperature was between 95° and 96°, but in most instances it usually did not go below normal. There was a very close relationship between the feeding of the patient and the development of these subnormal temperatures. Those who were kept long on liquid diet became very weak and their temperatures showed a decided tendency to become subnormal. Those who were given more solid food at an early stage showed but little such tendency.

As will be seen in the foregoing study of the symptoms, diagnosis is by no means easy, and there is a rather extraordinary variation in the symptoms as described by different men. The temperature curve is more often misleading than otherwise. Enlargement of the spleen, rose spots, and nose-bleed are also often absent. The presence of diarrhœa, with a peculiarly offensive odor, may be of some value when associated with other symptoms, and Lautre says it is absolutely diagnostic, yet it may, of course, be present in many other conditions. The diazo reaction in the cases presented here proved to be of no great value. Its frequent absence and the fact that it may be due to so many conditions other than typhoid fever make it of little use. The one sign which was altogether satisfactory was the Widal reaction. Without it, I feel sure that many cases of typhoid fever in old people must pass under other names.

As one would expect, the death rate is much higher than in younger individuals. Murchison in a series of collected cases found a mortality of 34.94 per cent. Of my 27 patients 6 died, making a mortality of 22.22 per cent. The causes of death were as follows: Intestinal hemorrhage, perforation and peritonitis, inanition and organic heart disease, phlebitis, pneumonia, and typhoid meningitis.

For lack of time I will say nothing of the postmortem findings, except that they conformed quite closely to the conditions found in younger people, with this addition, that many of the organs showed chronic disturbances as well as acute pathological conditions.

The treatment in most respects was such as would have been employed in younger individuals, with special watchfulness for cardiac, pulmonary, and genito-urinary complications. In the milder cases very little is needed to combat the fever, as it seldom rises high. Two of my patients had no treatment at all for this condition. For the more serious cases bathing of some sort was necessary. In some cases tub baths were borne quite as well as in younger people, but in a number of instances the patients shivered so excessively after the bath and became so cyanotic that we did not employ it at all in 7 instances, using sponge baths instead; six patients began with tub baths, but were treated later with sponge baths only, and 12 patients had tub baths exclusively. For the diarrhœa intestinal astringents and antiseptics were found beneficial, and for the bladder disturbances urotropin was unusually satisfactory. In those whose bladders were healthy at the onset of the disease, 7 grains of urotropin every four hours or at less frequent intervals almost invariably produced a favorable result in two to four days. In those who had chronic bladder troubles the results were less satisfactory. To prevent pulmonary complications during convalescence, I think it advisable to have the patient sit up in bed as soon as possible, and as already stated, something more than liquid diet should be given as soon as the fever has subsided to near normal. The dangers in old people of an exhausted condition with prolonged convalescence are, in my opinion, greater than are those of relapse and perforation from too early feeding.

ATROPHY OF THE PARATHYROID GLANDULES AND OTHER GLANDULAR STRUCTURES IN PRIMARY INFANTILE ATROPHY.

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My purpose herewith is to emphasize the constant finding of atrophy of the thymus gland in infantile atrophy, and to report a particular case in which there was marked glandular atrophy not limited to the thymus; but more especially I desire to call attention to the condition of the parathyroid glandules in infantile atrophy, to which, up to the present time, scant attention has been paid.

That certain glands are involved in disturbances of nutrition was pointed out as early as 1858 by Friedlieben,[1] who stated that the size and condition of the thymus was an index to the state of nutrition of the body. This observation has been confirmed by a number of investigators, among the more recent of whom may be mentioned Mettenheimer,[2] Stokes, Ruhräh, and Rohrer,[3] and Dudgeon.[4] The main conclusion of these authors may be summarized in the statement that atrophy of the thymus gland in children and wasting of the tissues go hand in hand. Dudgeon states, moreover, that atrophy of the thymus gland is the most characteristic morbid lesion found in cases of marasmus.

The average weight of the thymus at birth, as given by different authors, is at great variance, as will be seen from the following series of weights given in different text-books: Testut (Quain's *Anatomy*), 5 grams; Robinson (Morris' *Anatomy*), 14 to 28.5 grams; Vierordt (Hektoen's *Pathology*), 24 grams; Howden (Gray's *Anatomy*), 15 grams; Osler (*Practice of Medicine*), 14 grams; (second year, 25.30 grams); Ziegler (*Text-book of Pathology*), 24 grams; Roberts (Allbutt's *System of Medicine*), 15 grams; Fowler (*Encyclopedia Medica*), 15 to 30 grams; Friedlieben, 13.7 grams;

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[1] Die Phys. der Thymusdrüse in Gesundh. u. Krankheit, Frankfurt, 1858. Ref. Dudgeon (loc. cit.).

[2] Jahrb. f. Kinderheilk., 1897, lxvi, 55.

[3] Amer. Jour. Med. Sci., 1902, cxxiv, 817.

[4] Jour. Path. and Bact., 1905, x, 174.

two years, 26.2 grams. It appears that most writers have agreed with Friedlieben, and quite recently Hammer[5] gives the weight of the thymus at birth as 13.26 grams; and from the first to the fifth year, 22.98 grams. On the other hand, Thursfield[6] finds the average weight 6 to 7 grams, Dudgeon gives the weight as 7 to 10 grams, and Bovaird and Nicoll,[7] who recently reported on some 600 autopsies, give the average weight as 7 grams. In 25 cases, from birth to one year of age, excluding marasmus, I have found the average weight of the thymus to be 7.25 grams.

It seems to be a commonly accepted idea that the growth of the thymus gland proceeds during the first two years of life, and from that time remains somewhat stationary (although the parenchyma is replaced more or less by connective tissue) until puberty, when atrophy becomes rapid. Remnants of the gland can be found, however, in most instances in adults, even in advanced age. Hammer finds constant increase in weight up to puberty. Boviard and Nicoll state that the thymus does not increase in weight after birth; that there is no evidence of growth, although under certain conditions the gland may undergo hypertrophy. I am inclined to agree with them, and I have found no evidence to support the commonly accepted idea of growth of the gland up to two years of age. On the contrary, evidences of atrophy can frequently be found from birth, although these atrophic changes are not of necessity accompanied by loss of weight.

While a direct ratio has been noted between atrophy of the thymus gland and wasting diseases in children since the time of Friedlieben, as stated above, it is more recently that attention has been drawn to the fact that the most characteristic and constant lesion of the so-called primary atrophy in children, or marasmus, aside from atrophy of fat and muscle, is atrophy of the thymus gland. Marasmus, as defined by Morse,[8] "is a morbid condition of infancy in which there is extreme wasting of the soft tissues of the body without demonstrable organic lesions. It is the expression of continuous insufficient nutrition, due probably to defective absorption or assimilation rather than to defective digestion." Parrot[9] first described the disease in 1877 under the name "athrepsia," and considered it an acute starvation. Babinsky[10] found a chronic intestinal catarrh in his cases, with extensive atrophy of the intestinal wall, and concluded that "marasmic atrophy of the intestine caused death by starvation." This author also found that 37.1 per cent. of nutritive substances were unassimilated in this disease and passed in the feces. Bendix,[11] however, found Babinsky's nitrogen estimate exaggerated. Fenwick[12] found inflammatory changes in the mucosa of the stomach and intestines in 16 cases, and called attention to the lack of HCl in this condition. Heubner and Rubner[13] concluded that the disease is due to auto-intoxication from decomposed food in the intestinal tract, and Marfan[14] claimed a lessened activity of nutritive ferments to be the main cause of the condition. Symes[15] considered that "improper food induced disorganization and morbid secretion of the digestive glands."

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[5] Arch. f. Anat. u. Phys. Jahrg., 1906, Anat. Abt., Sup. Band, S. 91.

[6] St. Barth. Hosp. Rep., London, 1903, xxxviii, 129.

[7] Arch. of Pediat., September, 1906.

[8] Med. News, 1901, lxxix, 409.

[9] Progres medicale, 1874-5.

[10] Brit. Med. Jour., 1899, 1084.

[11] Jahrbuch f. Kinderheilk., 1896, Band lxiii.

None of these statements has lead to definite conclusions, however. Holt[16] does not consider the intestinal lesions described by other investigators as of importance in the etiology of the disease, and Wentworth[17] states that metabolism observations in infantile atrophy have shown a diminished power of absorption by the intestine for artificial foods, but they do not explain the cause of this failure to absorb, and he believes that the cause of infantile atrophy cannot be discovered by metabolism investigations.

Whatever the cause of the disease may be, the autopsy findings in this condition are unsatisfactory, and there is no lesion that is specific. At the postmortem, marked wasting of fat and muscle is noted; the heart, kidneys, and liver may show fat; the mucous and submucous coats of the stomach and intestines may show atrophy; the lymph nodes may be enlarged. Death may be due to a terminal infection, such as bronchopneumonia or enteritis. As has recently been emphasized, in addition to these findings there is present notable atrophy of the thymus gland, which is not specific for primary atrophy, but is found in other wasting diseases of infants.

Stokes, Ruhräh, and Rohrer examined 18 cases of marasmus and found the average weight of the thymus for 16 of the cases to be 2.2 grams. The smallest weighed 1.2 grams, and the largest weighed 7.5 grams. Dudgeon examined 15 cases and found the average weight 2.68 grams. The smallest weighed 0.905 grams and the largest, 5 grams.

I have had the opportunity to examine the thymus in 20 cases of marasmus at autopsies at St. Ann's Asylum, in infants from birth to one year of age. In every case I have found a notable atrophy of the thymus gland. The smallest weighed 1 gram (2 cases); the largest weighed 4.75 grams. The average weight of the gland was 2.472 grams. This result was practically the same as that of Stokes and of Dudgeon.

Little can be added to the histology of these glands as described by the aforesaid authors. I have found Mallory's phosphotungstic acid-hematoxylin stain the most satisfactory for their routine staining. With this stain the connective tissue is clearly differentiated, the cell nuclei are sharply defined, Hassal's corpuscles are prominently shown, degenerative products such as droplets of hyaline are distinct, and fibrin threads, or masses, which are frequently found in the bloodvessels, are beautifully demonstrated.

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[12] Brit. Med. Jour., 1896, ii, 829.

[13] Zeit. f. Biologie, 1899, Band xxxviii.

[14] Rev. mensuelle des mal. de l'enfance, 1901, tome xix.

[15] Brit. Jour. Chil. Dis., 1906, iii, 290.

[16] Diseases of Infancy and Childhood, 3d ed., 1907, 240.

[17] Jour. Amer. Med. Assoc., 1905, xlv, 771.

In general it may be said that in most instances all differentiation between cortex and medulla of the gland is lost. The replacement of lymphoid cells by cells which are evidently of connective-tissue or endothelial origin, as observed by Dudgeon, is frequently found. There is always a marked increase of connective tissue in the gland which divides it into numerous small lobules, some of which may be further invaded by connective tissue replacing the parenchyma. Hassal's corpuscles are rendered prominent by atrophy of the contiguous tissue, and present a variety of degenerative changes. The walls of the bloodvessels may be thickened. The vessels may contain fibrin thrombi.

It may be said in summarizing these cases that all infants dying of marasmus exhibit marked atrophic changes of the thymus gland.

While the thymus gland occupies a somewhat anomalous position in respect to other glands, nevertheless it cannot be wholly separated from, and usually is connected with, that group of structures that have to do with internal secretion, and are known as the "ductless glands." The structures included under this title of "ductless glands" are, at the present time, according to Vincent, the thyroid, parathyroids, cortical and medullary suprarenal, pituitary, the carotid and coccygeal bodies, and possibly the thymus.

In consideration of the physiological relationship between the thymus and these other structures, the following case[18] is of interest in showing that glandular atrophy is not always limited to the thymus in these cases of infantile atrophy, but may be exhibited in other glands of this group. In this instance, in addition to changes in the thymus, extreme atrophy was found in the thyroid, parathyroids, and medullary suprarenals.

This infant was admitted to St. Ann's Asylum when nine days old, and was in good condition. There were no errors of development. The weight was 9 pounds, 5 ounces. The infant progressively lost in weight, although at times there was a slight gain in weight which was never permanent. When the infant was seven months old it weighed 3 ounces less than when admitted. At this time physical examination showed the lungs clear, and the heart normal. It presented the typical picture of marasmus. During the next three months there was some improvement. At the eleventh month the infant weighed 10 pounds, 5 ounces. Then diarrhœa and vomiting developed and death ensued.

The autopsy protocol is, in brief, as follows: Weight, 10 pounds;

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[18] I wish to express my indebtedness to Dr. J. M. Brady for supplying me with the clinical notes on this case.

body is much emaciated; lividity of dependant parts; typical marasmus facies; no "rosary" or other evidence of rachitis; skin dry; muscles pale and atrophic; abdominal cavity normal; intestines somewhat distended; no mesenteric, abdominal, or retroperitoneal fat; appendix normal; mesenteric lymph nodes not enlarged; pleural cavities and pericardial cavity normal. Heart normal in size; epicardium, myocardium, and valves normal; foramen ovale, patent 2 mm. Lungs: upper lobes pinkish-gray in color; air containing throughout; normal on section. Lower lobes, dark red in color; firm, and present areas of solidification, especially in posterior part. On section color dark red, with irregular grayish, granular, slightly elevated areas throughout. Bronchi contain mucus, streaked with purulent material. Spleen normal in size, dark red in color, consistence fairly firm; normal markings are seen on section. Stomach, intestines, and pancreas normal. Liver slightly smaller than usual; color dark reddish-brown, consistence normal, markings normal on section. Gall-bladder and ducts normal. Gall-bladder filled with thin pale fluid. Kidneys normal in size, capsule strips easily leaving a smooth surface. Organs appear normal on section. Suprarenal glands, very pale grayish-white in color; the left measures 3 cm. long by 1 to 2 cm. wide, by 0.5 to 0.7 cm. thick. On section, cortical portion appears as dirty gray zone; medulla is brown. Right adrenal, 2.5 cm. long by 1 cm. wide, is very thin (1 mm.) and presents on section no color differentiation. Genito-urinary tract normal. Organs of neck: thymus, weight 1.25 grams; measures 2.5 × 1 × 0.5 cm.; has an accessory lobe 1.5 × 1 × 0.5 cm. Thyroid very small and pale; lateral lobes measure 1.5 long by 1 wide by 2 to 0.5 in diameter; weight of thyroid, 1 gram. Parathyroid glandules: the upper glandules are found in normal situation; they measure 2 mm. in longest diameter; the lower bodies are not found. Head not opened.

Microscopic examination of organs:

Heart negative. *Lungs:* sections from posterior part of lower lobes show areas where the alveoli are filled with an exudate consisting chiefly of polymorphonuclear leukocytes, with a few red blood corpuscles and desquamated alveolar epithelium. The capillaries of the alveolar wall are congested and tortuous. The smaller bronchi are filled with polymorphonuclear leukocytes. The larger bloodvessels are injected. In some sections collapsed alveoli (areas of atelectasis) are seen. *Liver:* the liver parenchyma presents a normal appearance; the liver cells do not show vacuoles; capillaries show slight injection in places; periportal connective tissue normal; Scharlach R. stain shows a small amount of fat, in fine droplets, in the liver cells. Spleen negative. *Kidneys:* the tubular epithelium is somewhat swollen and glandular; the glomeruli show a slight increase of nuclei; the bloodvessels are injected; walls of the bloodvessels show slight hyaline change. *Intestine:*

thin-walled; both mucosa and submucosa show noticeable atrophy; the lymphoid tissue is prominent. *Epiphysis of rib:* shows fairly regular, even line of calcification; there seems to be a slight excess of osteoid matrix at the extreme edges.

Thymus: The entire thymus was cut in serial sec ion and examined throughout, especially for inclusions of parathyroid tissue. None was found. In general the gland shows thick connective tissue stroma dividing the gland into small islets of lymphoid tissue, a considerable portion of which is being replaced by fatty connective tissue. Areas of focal necrosis also are seen where cells have lost their distinct outline, and a more or less homogeneous eosin staining mass results, in which swollen and fragmented nuclei can be seen. Hassal's corpuscles are prominent; they vary in size and present granular and hyaline degenerative changes. The bloodvessels are injected, and some show hyaline thickening of the vessel wall.

Left Suprarenal: The cortical portion is of about normal thickness, and shows fairly regular zonal differentiation, and presents no change worthy of note. The medullary portion, however, is much thinner than usual, and in a part of the gland is wholly absent. There is no characteristic cell grouping, but the cells are crowded together in closely-set masses, between which run irregular strands of connective tissue. The cells are swollen, the protoplasm granular or absent, and the cell boundaries not defined. In one place a zone of hemorrhage is seen between cortex and medulla.

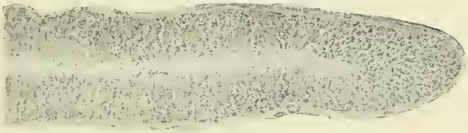

Fig. 1.—Sketch of the suprarenal body, showing replacement of medulla by connective tissue. (Magnified 60.)

Right Adrenal: Cortical portion similar to left adrenal. The medulla has entirely disappeared throughout and is replaced in part by a thin line of connective tissue (Fig. 1).

Thyroid: The gland shows a considerable amount of connective tissue stroma both between groups of tubules and separating the individual tubules (Fig. 2). The lining epithelium of the tubules is in great part detached from the basement membrane and the cells are heaped up irregularly, in places filling the lumina. There is no colloid present.

Parathyroid Glandules: The structure in general corresponds to the picture seen in the normal glandule. The cells form together hanging masses separated by connective-tissue stroma, bearing bloodvessels which are well injected. Within the individual masses the capillaries are found filled with blood. The "pallisade" or "balcony" cells are not seen at the edges of the gland. There is only one type of cell to be distinguished in the parenchyma. The cell boundary, which is usually distinct, is not to be made out in general. The cytoplasm of adjacent cells is more or less fused together, and in places is absent entirely. Many of the cell nuclei are swollen and irregular (Fig. 3). These degenerative changes are evident in varying degrees everywhere throughout the glandules. Scharlach R. stain shows absence of fat.

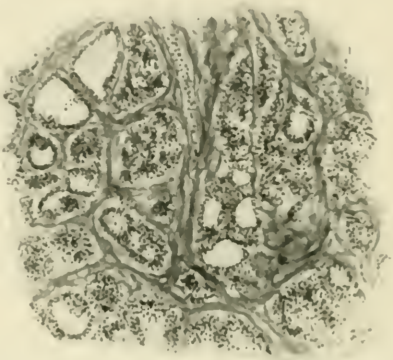

FIG. 2.—Thyroid gland, showing replacement of tubules by connective tissue and irregularity of the epithelium. (Magnified 105.)

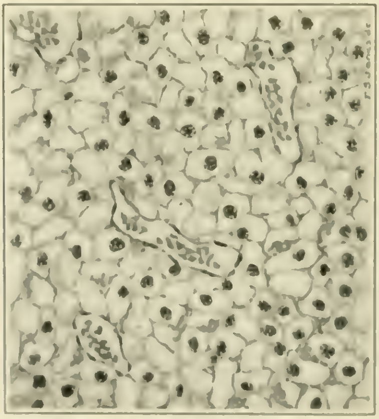

FIG. 3.—Parathyroid glandule from a case of infantile atrophy, showing degeneration of the principal cells. (Magnified 575.)

We have here, in brief, a case of infantile atrophy of one year's standing, in which the infant was kept alive for a considerably longer period of time than is usual in such cases. The immediate cause of death was a terminal infection (bronchopneumonia), which is not uncommon in these cases. The principal histological change, aside from the atrophy of fat and muscle tissue, was marked atrophy of the medullary adrenals, thymus, thyroid, and parathyroid glandules, which was so extreme as to attract attention macroscopically. The striking picture in this case can be accounted for, perhaps, by the long duration of the disease (one year), which lead to changes which were more marked than those usually seen in the cases, which run an average course of two or three months.

On account of the findings in this case it was thought desirable to see if similar atrophic changes could be found in these glands in other cases, and the following is the result of the study of the parathyroid glandules in 12 cases of infantile atrophy. (A detailed study of the thyroid, medullary suprarenals, and other glandular structures is purposely omitted in this paper.)

Parathyroid Glandules. The situation, general appearance, and microscopic structure of the parathyroid glandules of the infant do not differ essentially from that of the adult. The glandules are much smaller in the infant than in the adult. They average about 2 to 5 mm. in diameter normally in the infant, while in the adult they average about 8 mm. in longest diameter. Sandstroem[19] gives the adult length as 2 to 15 mm. They are more difficult to find in the infant than in the adult, not only on account of their small size, but owing to their resemblance to certain lenticular bits of tissue which extend upward along the posterior border of the thyroid on both sides of the œsophagus. This tissue is continuous with the interscapular gland of Hatai,[20] and at a later period of infant life is not to be distinguished macroscopically from fat, although Warthin[21] and others consider the structure a part of the hemolymph system. Bonnot, in a paper not yet published, but to which he has kindly allowed me to refer, considers also that this structure has to do with the formation of blood in the embryo, and to some extent in the adult. In marantic infants, in whom there is no fat, this tissue persists, and the tiny nodules are extremely difficult to distinguish in size, color, and situation from the parathyroids. By preserving the neck organs entire in Pick's solution I was finally able to get a color differentiation that distinguished this tissue from the glandules. Another source of difficulty is in distinguishing the parathyroids, especially the lower, from remnants of the thymus gland which are more much frequently found in the infant than in the adult. This in many instances can only be done microscopically. Several times I have found what macroscopically I presumed to be parathyroids, to be, microscopically, parathyroid incorporated in a remnant of thymus. This is to be expected from the close embryological relationship of the two structures, and Verebely has noted the same thing in the adult.

Microscopically, certain differences between infant and adult parathyroid may be noted. The most important seems to be that there is only a single type of cell in the infant glandule. The differentiation into "principal" and "functional" cells, as described for adult parathyroids, cannot be made. From other work on these glandules, not yet published, I had concluded that there is only one type of cell primarily in the parathyroid, corresponding to the "principal" cell, and that the so-called "functional" cell represents a modification of this cell, due either to degeneration or hyperfunction. Verebely[22] has also arrived at a similar conclusion. Fat, which is always present in the adult parathyroid, is not normally present in the infant parathyroid. Benjamins[23] has previously noted the increased amount of fat in adults as compared with the fœtus.

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[19] Ref. Schmidt's Jahrb., 1880, clxxxvii, 114.

[20] Anat. Anzeig., xxi, 369.

[21] Amer. Jour. Anat., 1901, i, 63.

The following observations on the parathyroids were made in 12 cases of marasmus, in infants from birth to one year of age. To these was added 12 control cases, from routine autopsies, other than marasmus in infants of the same age.

My attention was first called to the condition of these glandules in marasmus by the greater difficulty in finding them in the marasmic than in the other cases, and estimation of the weights and measurements showed that there was a considerable difference between the two groups of cases. The average measurements of the glandules in the cases of marasmus was 2 mm. in longest diameter for the superior, and 3 mm. for the inferior bodies. This is a little more than half the average measurements of the control cases.

The literature as regards the anatomy and histology of these bodies has been so thoroughly reviewed of late that it will only be referred to casually in this place. The number and position of the glandules showed the same variation in both series that is usually found in examining adult parathyroids. The position in general showed less variation than in the adult, possibly owing to the fact that if the glandules are removed some distance from their normal situation in the infant they may not be found.

It was impossible to obtain an exact table of the weights and measurements of these bodies, as some of the pieces that were apparently glandules macroscopically in both series proved microscopically not to be parathyroids, and deductions had to be made as nearly as possible by estimation. The estimate, which is as nearly correct as could be obtained under the conditions, showed in the 12 cases of marasmus the average weight of the four glandules to be from one-half to two-thirds the average weight of the four glandules in the cases not marasmus.

Individual glandules in the marasmus cases were found weighing as little as 0.06 gram (about a grain). None of the non-marantic cases showed such small size. In certain instances individual glandules were found in the control cases that were as small as certain of the larger glandules in the cases of marasmus.

The finding of the four parathyroids in each case in these series less frequently than in adult cases, is accounted for by the difficulties previously mentioned and the extreme smallness of the glandules. In the 12 cases of marasmus 4 glandules were found in 6. Three were found in 5 cases. In 1 case only 2 were found. In the 12 routine cases, 4 glandules were found in 8; 3 were found in 1 case; 2 were found in 2 cases, and only one glandule in one instance.

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[22] Virch. Arch., 1906, clxxxvi, 80.

[23] Ziegler's Beit., 1902, xxxi, 143.

Microscopically, the parathyriod glandules present, in the routine cases, a fairly uniform picture. They consist of closely set cell masses arranged in groups or in strands, which are separated by a rather delicate connective tissue stroma bearing bloodvessels. The relation between parenchyma and stroma is more uniform, and there is less tendency to form varying types of arrangement than in the adult. The cell masses consist of cells a little larger than those of the thyroid, which vary in size and shape and intensity of nuclear and cytoplasmic staining, but which conform to a single type, the so-called "principal" cells of the adult glandule. The cytoplasm of these cells may be indistinct or lacking, but the cell membrane is usually distinct. Along the capsule and septa these cells frequently assume a radial arrangement, the so-called "pallisade" formation. As previously noted, the so-called "functional" cells of the adult do not appear in the infant gland. Not infrequently bits of thymus tissue are found in contact with the glandules (Figs. 4 and 5).

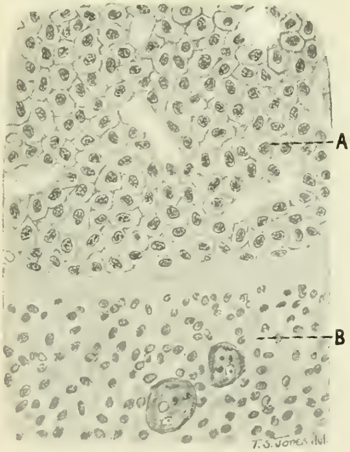

Fig. 4.—Normal infant parathyroid (*a*) in connection with (*b*) remnant of thymus gland. (Magnified 575).

In the cases of infantile atrophy the general structure of the glandule is the same as in the other cases. The changes that are found in this condition can be quite sharply differentiated into two types, which may be designated as (*a*) degenerative, and (*b*) sclerotic.

In the first type (Fig. 3) the glandules may be diminished in size or may be of normal size, and are pale in color, or, as in 1 case that showed intense congestion, the glandules were cherry red (Fig. 6). Microscopically, no increase of connective tissue is found, but the epithelial cells show everywhere a marked degeneration. They are larger and more irregular than normal, the nuclei are swollen and the cell boundaries are usually thickened, appearing as though the protoplasm had condensed in part at the cell peri-

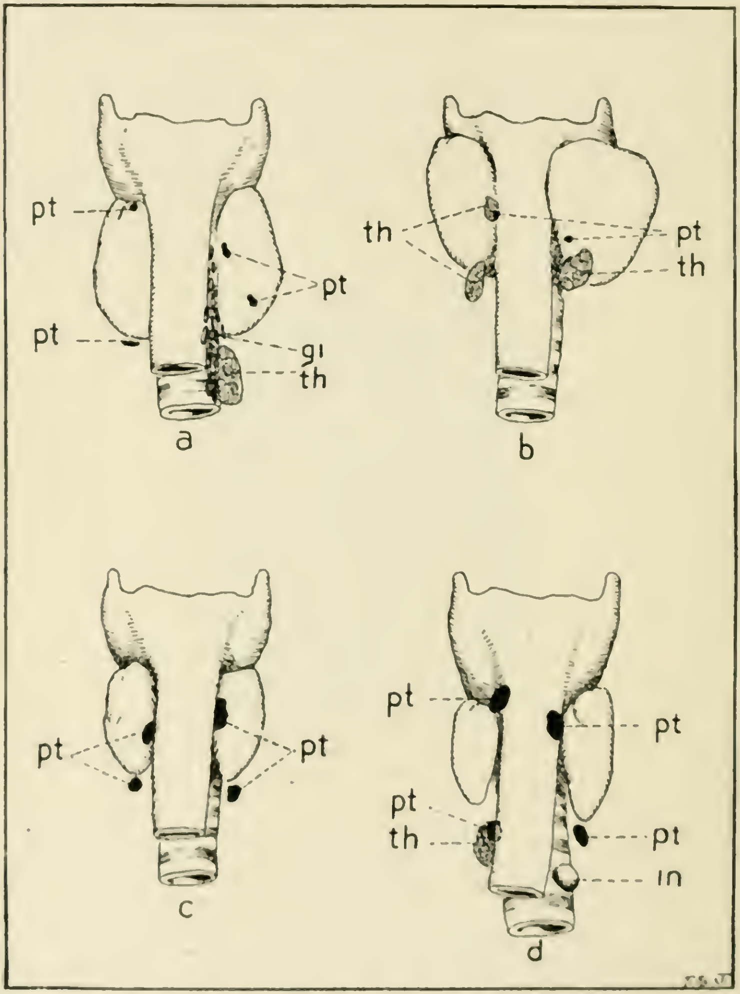

Fig. 5.—A and B. Sketch of the neck organs from cases of primary infantile atrophy, showing the small size of the parathyroid glandules, compare with C and D, which are control cases of infants of the same age. (*pt*) parathyroids, (*th*) remnants of thymus gland (in B replacing the two lower parathyroids and surrounding the left upper); (*gl*) represents tissue continuous with the interscapular gland. (*ln*) lymph node.

phery, as happens in certain forms of reticular degeneration. In places there may be complete loss of cell structure with crowding

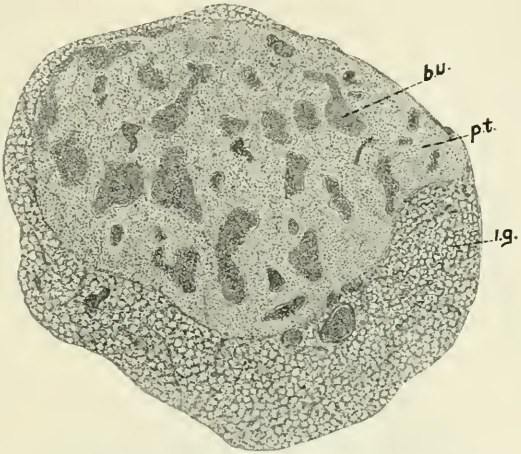

Fig. 6.—Extremely vascular parathyroid almost completely surrounded by tissue of the interscapular gland type. (Magnified 105.)

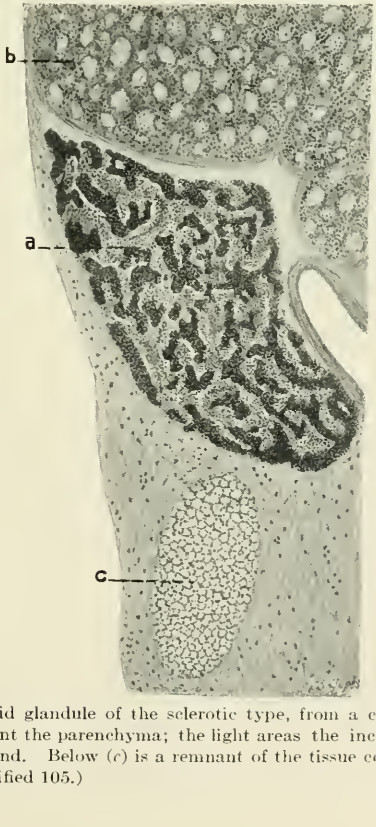

Fig. 7.—A parathyroid glandule of the sclerotic type, from a case of infantile atrophy The dark masses represent the parenchyma; the light areas the increased connective tissue. Above (*b*) is thyroid gland. Below (*c*) is a remnant of the tissue continuous with the interscapular gland. (Magnified 105.)

together of the cytoplasm into a fused mass, in which the nuclei are irregulary arranged. The bloodvessels may be greatly distended, but are usually moderately injected.

The most common finding, however, in these cases of marasmus is a marked increase in the connective tissue stroma of the glandules, which corresponds closely to the sclerosis (chronic fibrous parathyroiditis) described in the adult. These cases I have called, therefore, the sclerotic type. In these cases the glandules are smaller than normal, dark brownish-red in color, and firm. Microscopically the connective tissue stroma separating the cell masses is found increased in amount in varying degree (Fig. 7). The epithelial cells appear as irregular strands between the thickened bands of connective tissue. This connective tissue increase is frequently noted about the bloodvessels (Fig. 8). The connective tissue is loose, vascular, and rich in nuclei, which have a spindle shape. Mast cells are frequently seen in the connective tissue. In the epithelial islets between the connective tissue strands the "principal" cells are crowded together so that the typical sharp-lined epithelial structure is lost. The cell membrane is usually not seen, but masses consisting of closely set, irregularly arranged nuclei, without definite cell boundaries, are apparent. Groups of five or six cells occasionally in the centres of these islets preserve their original structure.

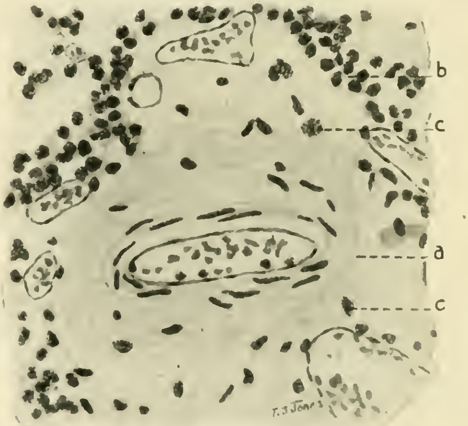

FIG. 8. Parathyroid glandule of the sclerotic type, high power, showing (*a*) perivascular increase of connective tissue, (*b*) crowding together of epithelial cells, and (*c*) mast cells in connective tissue. (Magnified 575.)

Briefly summarized, then, it may be stated that in primary infantile atrophy the parathyroid glandules show changes which may be degenerative in type, but which are, for the most part, progressive, and consist in replacement of a varying amount of the gland parenchyma by connective tissue stroma. These changes are similar in nature to those which are constantly present in the thymus gland in this disease. Moreover, these progressive changes are present in other ductless glands, and become especially apparent in long-standing cases of the disease, as evidenced in the extreme atrophy of the thyroid and medullary suprarenal in the cases here described.

It is by no means my intention to imply that these various glands are primarily at fault in infantile atrophy, but rather to emphasize the fact that failures of assimilation in this disease lead to wider changes than simple wasting of fat and muscle. The fatal result in this disease is more easily understood when we realize the constant involvement of structures that elaborate substances which are so essential to health, or even life itself, as are here found.

While no definite pathology has as yet been established for the parathyroid glandules, their great importance has been shown by a long series of investigators, beginning with those of Gley.[24] In many animals the removal of these glandules results in death, with tetanic symptoms, as has been shown by Gley, Vassale and Generali,[25] Welsh,[26] and many others. All results have not been in a cord, however, in the removal of these glandules in animals. The recent work of Vincent[27] and Jolly has shown that it is possible to remove both thyroids and parathyroids in certain animals without causing death.

In regard to the so-called tetania parathyroipriva that is supposed to accompany destruction of parathyroid tissue, and which is never present in infantile atrophy, the question might be asked whether such a condition might not be expected in cases in which the parathyroid glandules are involved, as here described. Vincent and Jolly, in their latest work on these glandules, are not able to confirm the theory that the removal of the parathyroids causes always severe acute nervous symptoms. They found that certain animals (rats, guinea-pigs, and badgers) did not suffer at all from their removal. Monkeys showed only transient nervous symptoms following the removal of these glandules, and nervous symptoms sometimes supervened when these bodies were left in situ after a neck operation. Therefore, we cannot consider by any means the fact wholly proved that in the human, the destruction of these bodies always induces tetany.

In the histological studies of parathyroids, MacCallum,[28] Banbrognini,[29] and Guizetti[30] have described changes in these glandules in gastric tetany, eclampsia, and traumatic tetany, respectively In general, however, the histological study of these glandules is negative in cases showing tetanic symptoms. I failed to find any alteration in these bodies in 9 cases of paralysis agitans, and in an infant dying in convulsions the glandules were negative.

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[24] Comptes rendus de la Soc. biol., 1891, p. 843.

[25] Revista di Patol. Nerv. e. Ment., 1896, i, 3 and 7.

[26] Jour. Anat. and Phys., 1898, 401.

[27] Lancet, August 11 and 18, 1906; Jour. of Phys., 1904, xxxii.

[28] Cent. f. alleg. Path., 1905, 385.

[29] Ref. Cent. f. allg. Path., 1907, xxviii, 82.

[30] Cent. f. allg. Path., xviii, 81. 1907,

There remains much to be known relative to the exact nature and function of these glandules and their real importance in the body economy. It is hoped that the preceding morphological notes may be of value in stimulating study of these bodies along other lines, and that this may throw some new light on the function of these structures, especially in conditions of impaired nutrition.

A TUMOR OF THE MITRAL VALVE.

By Leonard Blumgart, M.D.,
of New York.

(From the Pathological Institute of the Kaiser Wilhelm University at Strassburg, Prof. H. Chiari, Director.)

While making an autopsy (No. 111, April 2, 1907), I came upon a tumor of the left cusp of the mitral valve, which was given to me for purposes of examination.

The case was that of a woman, aged eighty-six years from the Home for the Aged. Disease of the heart was given as the cause of death. There was no clinical history.

The following are autopsy notes: A female body, 158 cm. long, well developed, and fairly well nourished. The brain is slightly œdematous, and its gyri atrophic. The lungs are œdematous, and the seat of a high grade emphysema. The trachea and bronchi show redness of their mucous membrane, which is also covered with a mucopurulent secretion. The peribronchial lymph glands are anthracotic and calcified. The kidneys are small, their capsules adherent, and the parenchyma shows degeneration. The other organs are normal, except the heart, which shows the following: (Fig. 1): The pericardium is smooth, and there is no exudate. The heart is enlarged and relaxed, weighing 495 grams. The right ventricle and right auricle are dilated, the former is slightly hypertrophied. The left ventricle is markedly hypertrophied, its wall being 2.5 cm. thick. The left auricle is also dilated. The endocardium is smooth. The myocardium shows no pathological change. The tricuspid and pulmonary valves are normal. The aortic valve is somewhat thickened, and in one place contains a calcified node. The aorta shows small areas of endarteritis deformans of a moderate degree. On the auricular side of the left or posterior cusp of the mitral valve is a tumor, 3.5 × 2.5 × 2 cm. in size, irregularly round, smooth, glistening, and softly elastic. It is pedunculated, the pedicle resting on the cusp, 5 mm. from its free edge. The rest of the cusp shows several slight aneurysmatic bulgings about 0.5 cm. in depth, whose convexities are upward.

The entire heart was placed in a solution of 100 parts of alcohol, 50 per cent., and 2 parts of formalin (Museum Preparation No. 3744). After hardening, the tumor was cut as indicated by the dotted line in Fig. 1. The cut surface of the right half of the tumor is shown in Fig. 2. The cut surface is seen to be a deep homogeneous brown red. A definite capsule is seen to surround the tumor, except at its inferior hollow ventricular surface. The capsule starts from the insertion of cusp, and passes over the tumor to continue into one of the tendons.

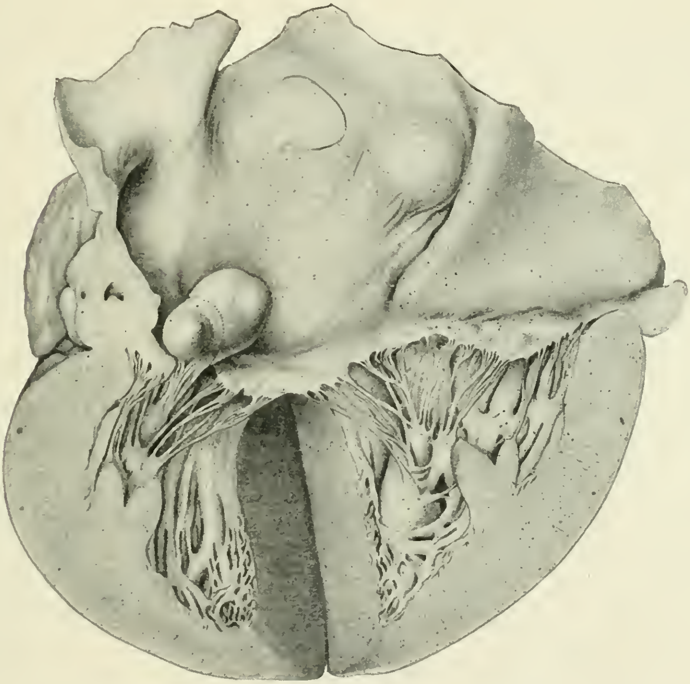

Fig. 1.—View of the interior of the heart showing the position of the tumor of the mitral leaflet.

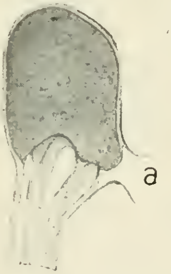

Fig. 2.—Cut surface of the right half of the tumor of the mitral leaflet (actual size).

A slice of the tumor was carried through alcohol, embedded in celloidin, and sections were stained with hematoxylin-eosin, hematoxylin-van Gieson, elastin (Weigert), hematoxylin-muci-carmin, muchematin, and thionin.

Microscopically, the tumor is composed of loose connective tissue, in whose meshes there is a large amount of granular brown blood pigment, most of which is situated beneath the capsule. Scattered through the fibrous tissue are areas of star- and spindle-shaped cells. These areas give a faint but positive reaction with muci-carmin and thionin, but are negative to muchematin. They also take a deep blue stain with hematoxylin-eosin. The blood vessels of the tumor are large cavernous spaces originating and entering the tumor from its ventricular surface; they subdivide, and branches reach to the capsule. The walls of the bloodvessels are lined with a single layer of flat endothelium and contain a fine layer of elastic tissue. No elastic tissue is found in the tumor. The capsule of the tumor, which varies in thickness, is composed of connective tissue, with a fine network of elastic fibers. Its outer surface is covered with a single layer of flat endothelial cells. The inferior hollow ventricular surface, which is without a capsule, is covered by a single layer of flat endothelial cells.

Tumors of the valves of the heart have been described by Curtis,[1] Debove,[2] Ribbert,[3] Guth,[4] Leonhardt,[5] and Reitman.[6]

My case corresponds with these in that it contains mucin-reacting tissue, fibrous, and elastic tissue. Following these observers, one would call the tumor a fibromyxoma. But the lack of capsule on its ventricular surface, together with the aneurysmatic bulgings on the rest of the cusp, inclines one to view this not as a new-growth, but as an organized thrombus in an aneurysm of the mitral valve in which the capsule around the thrombus is nothing but the dilated cusp.

Examining the literature as given by Leonhardt, I find no report of a similar condition. As there is a difference of opinion as to whether all fibro-myxomas of the heart, and especially of the left auricle, are true newgrowths or organized thrombi on the basis of a former endocarditis, I report the case in the hope that this and observations on similar cases in the future may help to clear up the question.

[1] Arch. de physiol. norm. et path., 1871–72, iv.

[2] Myxome pédiculé développé sur la valvule tricuspidale, Bull. de la Soc. anat. de Paris, 1873.

[3] Neumann. Ref. i. d. Deut. med. Woch., 1904, Lit. Beil. S. 9. Ueber Ruckbildung an Zellen und Geweben und uber die Enstheung der Geschwülste, Biblioth. med. Abt. C., 1897, Heft 9; Geschwulstehere, 1904, S. 233, ff.

[4] Prager med. Woch., 1898, Nr. 8.

[5] Ueber Myxome des Herzens insbesondere der Herzklappen, Virchow's Archiv, 1905, clxxxi, 347.

[6] Ein Fall von primaren Klappentumor des Herzens, Zeitschr. f. Heilk., 1905, Heft 1.

THE POSTERIOR MEDIAN PLEURAL BOUNDARY, WITH REFERENCE TO GROCCO'S SIGN.

By W. J. Calvert, M.D.,
OF COLUMBIA, MISSOURI.

The normal relationship of the posterior median boundary of the pleural cavities to the vertebral column, œsophagus, aorta, and heart is of importance in a discussion of the physical causes of the paravertebral dulness, or the so-called Grocco's sign.[1] In anatomical and clinical literature little or nothing has been said on this subject. In consequence I have undertaken a study of the easily accessible anatomical material in our laboratory at Columbia and have charted the results. I wish here to thank Dr. C. M. Jackson for the use of his material.

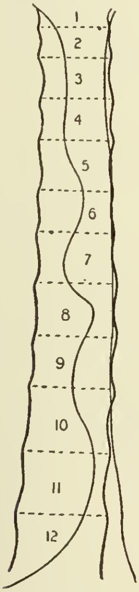

Fig. 1

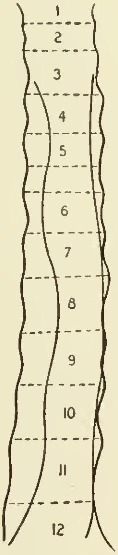

Fig. 2

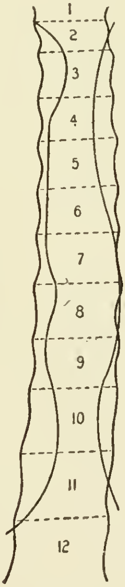

Fig. 3

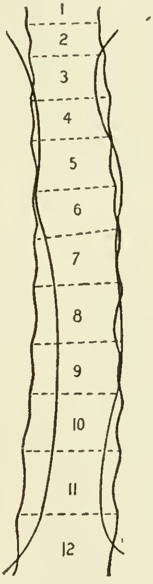

Fig . 4

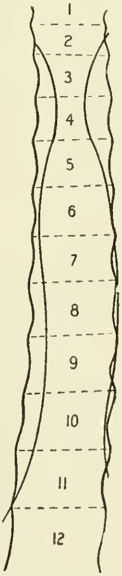

Fig. 5

The material considered includes 11 normal adults (Nos. 1 to 11); 1 newborn (No. 13); and 1 case of right-sided pleural effusion (No. 12). As the illustrations show clearly the course of both the right and the left pleural cavity, a description in detail is omitted.

The right pleural border in 11 of the 12 cases passes in front of the vertebral column varying distances often to the midline. In 1 case (No. 10) it extended entirely to the left of the column. The

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[1] See Thayer and Fabyan, Amer. Jour. Med. Sci., 1907, cxxxiii, 14.

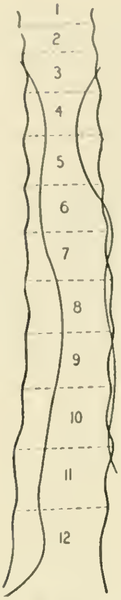

Fig. 6

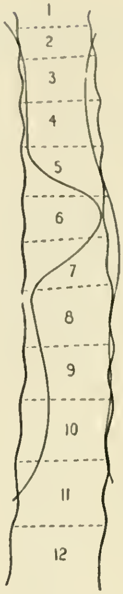

Fig. 7

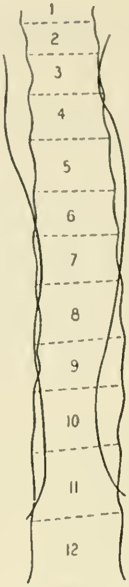

Fig. 8

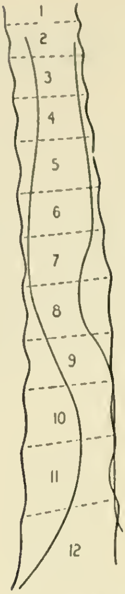

Fig. 9

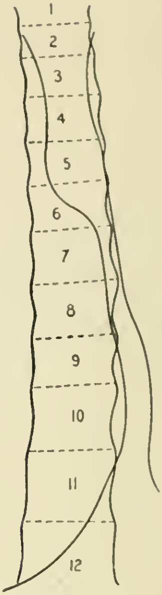

Fig. 10

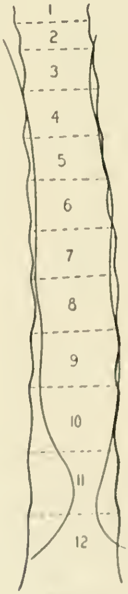

Fig. 11

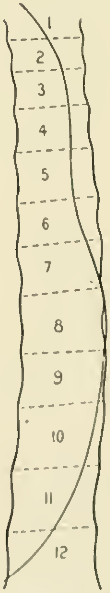

Fig. 12

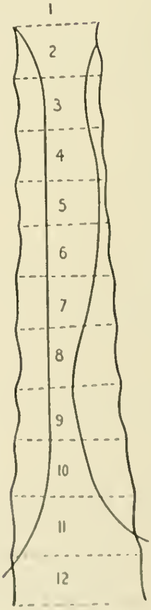

Fig. 13

Figs. 1 to 13 show a front projection of the dorsal portion of the vertebral column. The heavy lines represent the extreme limit of the right and the left pleural cavities.

Fig. 12 shows the border of the right pleural cavity in an old case of pleurisy with effusion. The degree of actual displacement to the left has not been determined.

Fig. 13 shows the pleural borders in a newborn. The relationship is the same as in adult life.

lower half of the line was further to the left than the upper. The left pleura did not extend on to the column to the same degree as the right most marked above and below, while the middle portion did not even touch the column. In practically every case the pleura was displaced toward the left by the descending aorta, especially from the fifth to the seventh vertebra.

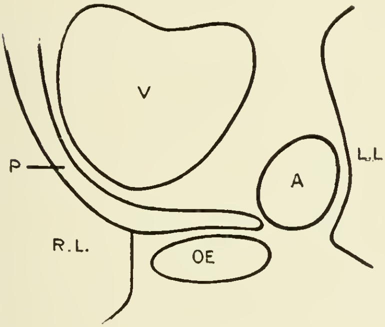

FIG. 14

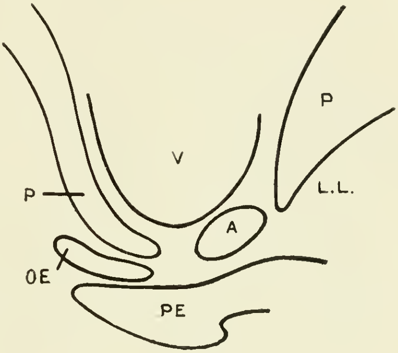

FIG. 15

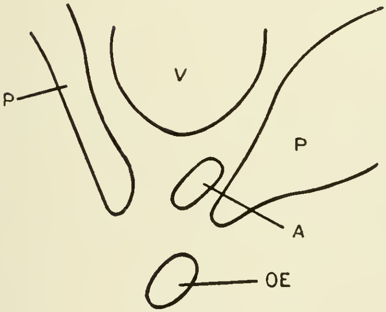

FIG. 16

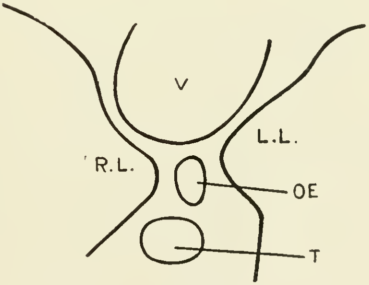

FIG. 17

The transverse sections show the relationship of the pleural cavities with the œsophagus, aorta, and heart, which is, of course, immediately in front of these structures. In Figs. 14, 15, 16, and 17 *P* represents the pleural cavity; *R.L.*, right lung; *L.L.*, left lung; *E*, œsophagus; *A*, aorta; *P.E.*, pericardium; *T*, trachea. Fig. 14 is a transverse section of Case 10 at the ninth vertebra. Fig. 15 is a transverse section of Case 2 at the disk of the seventh and eighth vertebræ. Figs. 16 and 17 are transverse sections of Case 13 at the ninth and fourth vertebræ, respectively.

THE DIAGNOSIS OF CEREBROSPINAL MENINGITIS BY CULTURES FROM THE BLOOD.

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AND

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At the present time the number of reported cases in which Diplococcus intracellularis meningitidis has been found in the general circulation is small—eight cases.[1] We report, therefore, the following case to illustrate the value of making a blood culture in all doubtful cases.

The patient, a female, aged fifteen years, entered the German Hospital May 5, 1907. The family history was negative. In the summer of 1906 the patient was in another hospital, where the diagnosis of acute articular rheumatism with acute endocarditis was made. She had a similar attack in the following December.

Five days before entrance to the German Hospital, the patient was suddenly seized with violent pains in the head, neck, arms, and legs, and she vomited repeatedly. These symptoms continued with more or less severity, except for the vomiting, up to the time of admission to the hospital.

On examination, she was a young girl of rather inferior development and poorly nourished. She was conscious of all that was going on about her. She seemed to be excessively tender all over her body, which fact rendered a satisfactory examination impossible. She would cry out whenever approached or touched. It was impossible to determine whether or not rigidity of the neck and Kernig's sign were present. The body was free from eruption. The area of the heart dulness extended from 2 cm. to the right of the sternum to the left midaxillary line. The apex beat was very diffuse, but was best seen in the sixth interspace. A whistling, systolic murmur was heard at the apex and was transmitted to the axilla. Except for the excessive tenderness and the heart condition, the physical examination was negative. The temperature was 102.4° to 103°; the white blood cells 34,800.

May 6, under aseptic precautions, a small incision was made over one of the superficial veins at the bend of the elbow and the vein exposed. The needle of an ordinary hypodermic syringe was introduced into the vein and 4 c.c. of blood was removed and distributed equally in two flasks containing 75 c.c. of sterile bouillon each. On May 7, the bouillon was found to contain a diplococcus which was isolated in pure culture on agar plates. The colonies were few and scattered and appeared round, flat, grayish, translucent, moist, and shining. The organism decolorized by Gram's method. The presence of a Gram decolorizing diplococcus which grew on agar with the characteristics named above lead us to make a diagnosis of Diplococcus intracellularis meningitidis. On the strength of these findings a lumbar puncture was done and 50 c.c. of fluid withdrawn, in which intracellular diplococci, decolorizing by Gram's method of staining, were found.

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[1] Sutton, Jour. Amer. Med. Assoc., June 8, 1907.

On May 11, the patient's opsonic index to the diplococcus obtained from the blood, was 100 per cent., and she was given an injection of 0.5 c.c. of a "personal vaccine" standardized of 20,000,000 to the cubic centimeter. On that date her temperature was 103° to 103.4°. The following day the patient's temperature declined, and she was much brighter and ceased to cry out when approached. On May 15, her opsonic index was 172 per cent., and her temperature was 101.4° to 101.8°. Her temperature continued to fall by lysis and her general condition improved steadily. On May 17, her temperature reached 99.8° where it remained with slight variations until the 25th, when it reached normal and thereafter remained normal. The recovery was otherwise uneventful.

REVIEWS.

The Johns Hopkins Hospital Reports. Volume XIII: Studies in Urological Surgery. By Hugh H. Young, M.D.; H. A. Fowler, M.D.; John W. Churchman, M.D.; John T. Geraghty, M.D.; Louis C. Lehr, M.D.; A. R. Stevens, M.D.; Stephen H. Watts, M.D., and F. H. Baetjer, M.D. Pp. 612. Volume XIV: Studies on Hypertrophy and Cancer of the Prostate. By Hugh H. Young, M.D., Associate Professor of Genito-urinary Surgery, Johns Hopkins University. Pp. 629. Baltimore: Johns Hopkins Hospital Press, 1906.

These volumes, printed on a great deal of paper, consist mostly of reprints of articles which have appeared during the last few years in the *Johns Hopkins Hospital Bulletin*, and in a few other journals. Many of the articles have been elaborated, and a few, we believe, are now published for the first time. These facts are nowhere clearly stated in the present publications, and the reader who was not already familiar with current surgical literature could only infer that some of the treatises here included had heretofore seen the light of day from the fact that the cases reported and the references to literature somewhat mysteriously cease in 1904 or 1905, although the volumes did not appear until early in 1907.

Volume XIII covers a wide range of topics dealing with genito-urinary surgery, including twenty-one articles in all. Among the more valuable contributions are: II. The possibility of avoiding confusion by B. smegmatis in the diagnosis of urinary and genital tuberculosis, by Drs. Young and Churchman. VII. The treatment of stricture of the urethra, by Drs. Young and Geraghty. VIII. The treatment of impermeable stricture of the urethra. IX. The use of the cystoscope in the diagnosis of diseases of the prostate—both by Dr. Young. XI. Chronic prostatitis, by Drs. Young, Geraghty, and Stevens; and XII. A modern method for the performance of perineal lithotomy, by Dr. Young. Although many of the other articles contain matter of much merit, the limited space allotted to a review prevents reference to them.

In their studies as to the possibility of differentiating between B. smegmatis and B. tuberculosis, Drs. Young and Churchman show (1) the uncertainty of the staining and cultural methods at present employed; (2) that irrigation, performed according to a certain technique devised by them, is absolutely efficient in clearing away all smegma bacilli, and (3) that all smegma-like bacilli which remain after this irrigation are truly the bacilli of tuberculosis. The technique employed is sufficiently simple, and they have had positive results in all the 104 examinations made according to this method.

Drs. Young and Geraghty, in their article on the treatment of stricture of the urethra, give some interesting and valuable statistics; but with all of their suggestions and conclusions few surgeons will agree. About 400 cases in all are analyzed. The results of gradual dilatation showed that of 17 patients who were traced after being treated more or less thoroughly, only 24 per cent. had recurrence; and 2 of these only after a period of eight years; whereas of 34 patients who returned for treatment during only a limited period, 67.7 per cent. had recurrence. Divulsion is condemned; internal urethrotomy is rarely employed, and then only for resilient strictures of the penile urethra. In 3 cases of impermeable stricture of the penile urethra, Dr. Young did perineal urethrotomy, and followed this by retrograde internal urethrotomy of the penile strictures. In 1 of these cases this method resulted in rupture of the urethra anterior to the strictures, necessitating external penile urethrotomy. The latter operation would probably have been quite satisfactory as a primary procedure; indeed we do not understand the delight the Johns Hopkins surgeons seem to take not only in doing a complicated operation when a simple would suffice, but also their passion for attacking strictures from the rear.

After perineal urethrotomy for strictures, they usually leave a catheter *à demeure* in the bladder, draining through the whole length of the urethra; in 2 cases only, was it allowed to emerge through the perineal wound; while in 9 patients no catheter was employed. In 1 case no instrument of any kind could be passed ten days after the operation, and another operation was done; it is not stated whether this unfortunate patient was one of those in whom the experiment was tried of leaving no catheter at all in the bladder. Among 79 operations there were 6 deaths; but as Dr. Young, with characteristic modesty, proceeds to explain, 4 of these were almost dead before operation, which reduces the mortality to 2.5 per cent.; while the remaining 2 deaths were really due to pre-operative conditions—the resultant of these forces being that about twelve pages later (p. 156) it is implied that *no patients* died after operation

Resection of the urethra was employed in only 2 patients, both traumatic cases: 1 patient, from whom 2 cm. of the bulbous urethra were excised with end-to-end anastomosis, was traced for seven years, and had then a urethra which admitted an 18 French sound —a remarkably excellent result. They urge, and we believe with propriety, that immediate perineal section be done in traumatic rupture of the urethra accompanied by much hemorrhage, even if there be no retention. We are not so sure that it will always prove wise to attempt "plastic repair of the injury" as a primary operation.

Certain statements in the article on impermeable stricture cannot pass unchallenged. That most surgeons are in the habit of abandoning perineal section (by which we mean external perineal urethrotomy without a guide) after a few aimless strokes of the scalpel, when they find it impossible, on exposing the anterior face of the stricture, to insert a guide into the urethral channel, and are then wont to attempt retrograde catheterism by means of suprapubic cyastotomy—is a statement which certainly does not describe the custom of most surgeons of experience in this city. Many facts tend to confirm us in our opinion that it will be immediately safer for the patient and more to his ultimate advantage, for the surgeon to stick to his perineal section and work it out on these lines if it takes all summer. But in saying this we do not wish it to be understood that we counsel the employment of so tedious an operation as perineal section, in patients who are acutely ill. For suchcases—patients with impermeable stricture and acute retention—we have at our disposal a procedure to which Dr. Young witheringly refers as "dangerous, inaccurate, unsurgical, and particularly prone to lead to injury of the rectum." In thus describing the old operation of Dionis, so brilliantly employed and so widely popularized by Mr. Cock, of Guy's Hospital (Dr. Young spells it Cocke), it is very evident that the distinguished professor of genito-urinary surgery is not well informed either of the nature of the operation itself, or of the indications to meet which it was proposed. Dr. Young speaks of it as an "incision into the prostatic urethra blindly from the perineum and retrograde catheterization through the urethrotomy wound in that location." He says that after failure to find the urethra by perineal section, and because of the dangerous, inaccurate, and unsurgical nature of "Cocke's" operation, he has been driven, on several occasions, to perform suprapubic cystotomy and retrograde catheterism. Now everyone—except perhaps Dr. Young—knows that Cock's operation was not proposed as a sequel to, but as a substitute for, perineal section; and when so employed, especially in patients with retention (for whom indeed it is best reserved), there is no other operation which will relieve such patients so swiftly, so safely, and so pleasantly. We admit that perineal section is one of the most difficult operations in surgery, and that the surgeon often passes axious moments until, as the urine begins to flow, the bystanders see a look of relief pass over his countenance; but why Cock's operation should be so crushingly condemned because it is not suitable for what it was never intended, we cannot understand.

Dr. Young proposes, and has adopted in one case, a form of operation, which, while undoubtedly of value in patients without retention, can never, we think, take the place of Cock's operation in acute cases. This consists in exposing the membranous urethra to full sight, as in his operation of perineal prostatectomy, opening it, and dividing the impermeable strictures from behind forward. This modification of the operation of John Hunter is certainly preferable to the suprapubic route, which Dr. Young considers the only alternative; and surgeons who are uable to dissect the urethra from before backward may be very glad to make the attempt to reverse the process.

One error in the volume, presumably a misprint, must surely be noted: the statement (p. 154) that Otis showed that the normal urethra frequently had a calibre of from 35 to 40 mm. Evidently what is intended is 35 to 40 French, or 35 to 40 *thirds* of a millimeter. Not even the enthusiasm of Otis could convince anyone that the normal urethra was ever over an inch and a half in diameter. Although this is one of the most glaring typographical errors, there are many others throughout the volume; indeed if the volume has received any editing at all, it is difficult to see in what it consisted.

Volume XIV contains only three articles: 1. Treatment of prostatic hypertrophy by conservative perineal prostatectomy. 2. Recto-urethral fistulæ. 3. The early diagnosis and radical cure of carcinoma of the prostate—all by Hugh H. Young, M.D. The text comprises less than 200 pages, the remainder—considerably over 400 pages—being composed of case histories, the records of 145 cases of perineal prostatectomy consuming no less than 334 pages. The object of this exhaustive analysis is, Dr. Young states, "to dissipate the false impression as regards the results in this country, and to remove the absolutely unjust, not to say false, idea of perineal prostatectomy which has been disseminated elsewhere." It will probably be news to many to learn that the results of prostatectomy in this country are regarded elsewhere as so deplorable; and the innuendo that a certain well-known British surgeon has been disseminating false ideas of the perineal operation, makes us earnestly hope that Dr. Young has not neglected to send him a copy of the present volume, bound in full morocco, with edges gilt.

Dr. Young's work is so well known, in this country at least, that extended mention of these articles is not necessary. The reviewer may be permitted to remark, however, that the great desideratum which a teacher should have in mind is not so much to impress his pupils with his individual skill and dexterity as to qualify them to go and do likewise. And those of us who sit humbly at the feet of the specialists, and watch with hungry eyes for the morsels which fall from their operating tables, note with regret the multiplicity of special instruments which Dr. Young now regards as indispensable to the proper performance of "his operation;" to wit: the cystoscope, the prostatic tractor, the new bifid retractor, a special posterior retractor, a special anterior retractor, and two special lateral retractors, not to mention the "enucleator" or special blunt dissector, the special lobe forceps, the special Halsted clamps to catch the urethral mucosa, and the Halsted operating table. We observe no mention of the best form of needle-holder for this operation, and presumably an ordinary scalpel is sufficient for most cases.

Among accidents which occurred during the operation, he mentions 4 cases in which false passages were produced by the *orderly* who held the staff in the urethra. Young complains of the trouble this made for the operator, but makes light of the disability caused the patient—in 2 of these cases only filiforms could be introduced after recovery from the operation; "but no definite strictures were produced" (p. 84).

Dr. Young's operative results are brilliant—he has now done 185 consecutive perineal prostatectomies, with only 7 deaths, a mortality of 3.7 per cent. It seems like painting the lily when he adds that in several of the fatal cases death was in no way caused by the operation; that in the last 100 cases there have been only 2 deaths, and that in the last 60 cases "there has not been a single death or bad result."

Perineal fistula persisted in 6 out of the 145 cases analyzed. The urinary functions were fully restored in all but 23 of the patients. It may be said, indeed, that only in about 14 per cent. of cases has the operation failed to give absolute relief, and that in all of these cases the postoperative condition is better than that which existed before the operation was done.

In closing it may be said that while Dr. Young is only presenting a study of his perineal prostatectomies, yet nowhere in all the volume does he even imply that there is the remotest excuse for the existence, still less for the adoption, of any operation other than that elaborated by himself. He refers to operations by any other technique as those done "blindly, blunderingly and barbarously, simply to save a little time." Yet what else than maudlin inconsistency, shall we call it, when he proposes, for the radical cure of recto-urethral fistulæ (7 cases of which have been encountered), an operation which has as one of its essential points suprapubic drainage of the bladder, which does away with "spasmodic efforts of urination"—thus tacitly admitting that suprapubic drainage procures rest for operative wounds in the region of the prostate.

Young's operation for radical removal of the prostate, neck of the bladder, seminal vesicles, and portions of the vasa deferentia has already been reported in the *Johns Hopkins Hospital Bulletin* (1905). He here records 4 cases of this operation, with "no operative mortality," although 1 patient "died six weeks after the operation as the result of an operative mistake." Even with a mortality of 25 per cent. (which Dr. Young is far from admitting), this operation should not hastily be condemned. Desperate diseases require desperate remedies; and if only the case reports convinced one that ultimate freedom from recurrence was assured, or that the patient's closing days were less distressing after the operation than if none had been performed, other surgeons would be more ready to adopt the extensive removal of the cancerous prostate advocated by Dr. Young.

A. P. C. A.

The Nursling: The Feeding and Hygiene of Premature and Full-term Infants. By Pierre Budin, M.D., Professor of Obstetrics in the University of Paris. Authorized translation by William J. Maloney, M.B., Ch.B., Fellow of the Obstetrical Society of Edinburgh; with an Introduction by Sir Alexander R. Simpson, M.D., LL.D., D.Sc., University of Edinburgh. With 111 diagrams in color, and other illustrations. Pp. 199. London: The Caxton Publishing Co., 1907.

This remarkable series of lectures by Pierre Budin, now for the first time presented to English readers through Dr. Maloney's translation, constitutes one of the most valuable contributions to the study of the nursing infant that has ever been published. Budin is one of the few obstetricians who has seemed to realize that the newborn infant is something more than an annoying but relatively unimportant complication of the puerperal state. More than this, he seems to have taken a conscientious interest in the welfare and progress of the babies born under his supervision, and in this series of lectures to his classes has presented a most exhaustive study of the nursing infant that deserves the respectful attention of every physician whose practice brings him in contact with helpless babyhood. In no single publication do we know of such a wealth of material illustrating all possible phases of the natural feeding of the infant. Of especial value is the admirable presentation of the care and feeding of the weakling—the premature or congenitally feeble infant weighing at birth less than 2500 grams (5 pounds)—that should prove a revelation to many practitioners of more than average experience in obstetrical work. To the pediatrist this section cannot fail to be of extraordinary interest, emphasizing as it does the value of constant records of the infant's weight and temperature as a guide to its progress and an infallible warning signal of its danger. The subject of wet-nursing is treated in masterly fashion, and its significant lessons should aid in saving many infant lives now ruthlessly sacrificed by careless obstetricians and incompetent nurses, to the latter of whom alone is too often entrusted the practical management of the important function of lactation, and whose one thought seems to be to get the baby upon a bottle as quickly as decency permits.

It is worthy of note, however, that Budin's experience has been gained among a class of patients with whom breast feeding is a normal and physiological result of maternity. Coming from the peasant and bourgeois classes, normality of infantile digestion is the rule, and it is safe to say that many of the difficult problems of infant feeding with which we of American experience are so familiar are practically unknown in Budin's clinic; furthermore, his patients, who are practically confined to those born in his wards, have not as a rule been subjected to dietetic experiments before coming under his care. For these reasons we believe his experience in artificial feeding lacks many of the difficulties with which American pediatrists are so frequently confronted. His rule for all cases of partial or complete artificial feeding is to give sterilized whole milk containing from 3.75 to 4 per cent. of fat, in proportions to be determined by the weight and the rate of progress of the infant. Dilution of milk or any of the finer modifications is in his experience rarely required.

Commenting on the results of his observations under this plan of feeding, Budin asserts that he is "absolutely unacquainted with the so-called 'undiluted milk dyspepsia.' White stools of the consistency of putty, the pathognomonic sign of the new dyspepsia, indicate merely that the infant is being overfed. We never see them, for we do not overfeed. It is rarely that we need to prescribe more than 1000 to 1200 grams of milk, even for infants in their second year."

"As for rickets, not a single case has occurred in any of my consultations since I first began in 1892. Neither will you see eczema among our little patients. Since 1892 I have observed only 2 cases, both of which were suckled by mothers indulging in excess in food and drink.

"As for the so-called 'infantile scurvy,' which is alleged to follow the use of sterilized milk,. . . .I am still looking for my first case."

Such statements from a man of Budin's skill and experience are sure to attract a lively interest upon this side of the Atlantic; and while we believe that the conditions of infant feeding with which we are familiar are in many instances less favorable than those in which Budin has labored, his experience in what may be termed normal cases deserves the attention and close study of our most experienced pediatrists. One statement of his which is significant in this aspect of the subject is this: "As regards artificial feeding from birth, my experience is yet too limited to warrant any dogmatic statement of the most advisable method during the first few weeks of life. As I always endeavor to insist on breast feeding, my cases are not numerous enough to base any definite opinion upon. Probably in those infants who do not tolerate pure milk at the beginning, some form of digested milk, such as that which I used for weaklings, might be found of service." This is a confession which after all may serve to explain what at first sight seems a revolutionary dogma; and we are forced to the conclusion that digestive weaklings are a much more common class with us than they seem to be among the bourgeois of Paris.

With this commentary we most heartily commend this monograph to the American profession, believing that our methods of infant feeding have not by any means received a death thrust, but that both French and American students of the gentle art of race preservation may have much to learn from an impartial study of each other's successes. T. S. W.

The Principles and Practice of Dermatology, Designed for Students and Practitioners. By William Allen Pusey, A.M., M.D., Professor of Dermatology in the University of Illinois; Dermatologist to St. Luke's and Cook County Hospitals, Chicago. Pp. 1020. With one colored plate and three hundred and sixty-seven text illustrations. New York and London: D. Appleton & Co., 1907.

In taking up a new systematic work on cutaneous diseases one naturally is disposed to ask certain questions the answers to which will in a measure stamp the calibre of the work. Thus: What classification of the diseases does the author adopt? What innovations in general upon established custom are made? What is the nomenclature, new or that in common use? Is the author under the influence of foreign thought, or has he produced a work based largely upon experience in his own country? Such questions are generally solved without effort by perusing pages here and there, the author showing his hand plainly to him who reads between the lines. In the case before us the author has long been known as a laborious student and practitioner in the field of dermatology, and as one who not only is seeking the truth, but who aims to give expression to it in his writings. We should, therefore, expect to find much of interest in a systematic work like the present, and in this we are not disappointed. To give a synopsis of the contents would be impossible, but some of the more striking features may be referred to.

Much thought has been bestowed upon the subject of the principles of dermatology. A correct understanding and appreciation of these principles really mean everything to the clinician, and distinguish the casual observer of skin diseases (of which class there exist everywhere too many) from the skilled practitioner who comprehends the etiology and pathology of the disease before him as well as its treatment. We commend all that Dr. Pusey says on this broad subject, and anyone who reads this part of the book will be repaid for the time spent. In the classification we do not think the matter has been quite so happily dealt with, for some innovations upon long-established lines of thought have been made which at first seem to be inconsistent with the foundation stone of the structure. Thus, there is a class of "inflammations," one of "angioneurotic dermatoses," another of "dry, scaly, inflammatory diseases," and still another of "infectious diseases." This classification may be practical and in one sense it is so, but the method of thus grouping cutaneous diseases is a departure from the lines upon which we hold true classification should be based.

But, to proceed, the general view taken of the whole subject of cutaneous diseases is in the main in accord with that of most teachers in this country. Diseases, common and rare, are described much as occurs in the standard works of the day, and everywhere may be observed an exposition, brief or lengthy, of the latest views upon etiology, pathology, and treatment. The subject-matter is thus brought well up to date. In the matter of treatment of some of the diseases we think more space might have been allotted to internal medication, diet, and the like. Thus, in the chapter on the internal treatment of eczema many more practically useful points (doubtless well known to the author) might with advantage have been elucidated. It is within the reviewer's experience to state that many excellent general practitioners not infrequently virtually ignore internal treatment in chronic eczema, with the result that the patients are neither cured nor benefited as they should be. The class of "dry, scaly, inflammatory diseases" includes lupus erythematosus, seborrhœic dermatitis, pityriasis rosea, psoriasis, lichen planus, dermatitis exfoliativa, and a few other less well-known diseases. As scaly eczema is not admitted to this class, and as lupus erythemotosus is usually regarded as being much more in its symptomatology than a scaly, inflammatory disease, this group does not seem to rest on a very solid foundation. In the class of "angioneurotic diseases" the most diverse cutaneous lesions occurring in different diseases occur, for here are found erythema multiform, herpes, pemphigus, pellagra, purpura, etc.

Considerable attention is bestowed on A. E. Wright's method of treating tuberculous and staphylococcic infections by the use of hypodermic injections of sterilized cultures of the bacteria, favorable results being reported in such diseases as furuncle, acne, and lupus vulgaris. In psoriasis, sodium cacodylate by the mouth in half-grain to one and a half-grain doses, thrice daily, has been found of value. The drug soon gives a garlicky odor to the breath, but does not readily disturb the stomach. The hypodermic method of administering arsenic is favored. Attention is directed to the not infrequent association of this disease with rheumatic and gouty states.

Among the new diseases described mention may be made of dermatitis vegetans, erythema elevatum, hydroa vacciniform, blastomycosis, and granuloma pyogenicum. Numerous other rare and foreign diseases are briefly described. It is gratifying to note the introduction of the eruptive fevers in the treatise. They assuredly belong here. The descriptions are brief, and, of course, with special reference to the skin. Many excellent photographs (mostly from Schamberg's collection) add greatly to the value of this chapter.

Scrofuloderma receives a special chapter, to which it is entitled, the author separating it from true tuberculosis. It might well have been longer, and made to include the small and the large pustular scrofulodermas. The former is described elsewhere, but the latter does not seem to be mentioned. The author prefers the French word "syphilide" to (it seems to us) the more correct and in every way more desirable term "syphiloderm." It has always seemed that in connection with the English language the French word syphilide is out of place, and this for several good reasons. The author very wisely cautions as to the indiscriminate or careless use of the Röntgen rays. With his large experience in the employment of this therapeutic agent, caution deserves heed. The high-frequency current has proved disappointing to him.

The illustrations are unusually profuse, and, speaking generally, are satisfactory, most of them being originals, and many from the author's collection.

In concluding, it may be stated that a wide field is covered in the book, and that for this reason the author's task has been by no means an easy one. The subject matter is so abundant and varied that the physician, we believe, will derive more profit from its perusal than will the student. There is much more in the volume than the student can possibly digest. The author may be congratulated on the part he has played in advancing our knowledge of cutaneous diseases to a higher plane than heretofore. We bespeak a very kindly reception for the book. It deserves it. L. A. D.

A LABORATORY MANUAL OF INVERTEBRATE ZOOLOGY. BY GILMAN A. DREW, Ph.D., Professor of Biology in the University of Maine; in charge of Zoological Instruction at the Marine Biological Laboratory, Woods Holl, Massachusetts. 201 pages. Philadelphia and London: W. B. Saunders Co., 1907.

REFERRING a laboratory manual of invertebrate zoology to a medical journal for review is suggestive of the growing interest of medical men in medical zoology and the relations of a large group of the lower animals to man in the matter of the production or transmission of disease. It is safe to predict that at no very distant date the work of instruction in our medical laboratories of bacteriology will be expanded to include a not inconsiderable amount of attention to the protozoa, as well probably to at least the medically important worms and arthropods and to various animals of higher types concerned in the transmission of human disease.

The volume in hand is intended as a laboratory handbook for beginners in invertebrate zoology and has no immediate application to medical problems, save as a medical man or student may care for a guide to laboratory study of these forms of animal life. It has been built up from a set of laboratory directions used in the instruction of the class in general zoology in the Marine Biological Laboratory at Woods Holl. Its especial value lies in the employment of the Huxleyan methods of study in the directions for observation, and in the questions for determination which are incorporated in the text under the various types set for study, thus not merely guiding in the systematic morphological study of types, but also stimulating an appreciation of the adaptations exhibited by these low forms. The classification followed is authoritative; the definitions modern and admirable; the instructions full and well adapted to student work, and the methods adopted simple and well selected. For use either by isolated workers or as a laboratory book in collegiate biological departments in the preparatory course leading to medicine, the book is to be strongly commended to favor.

A. J. S.

SURGERY OF GENITO-URINARY ORGANS. By J. W. S. GOULEY, M.D. Pp. 531. New York: Rebman Co., 1907.

THE author of this handbook discusses most of the subjects usually included in genito-urinary surgery. The volume is something more than a new edition of his monograph on *Diseases of the Urinary Organs*, published in 1873; chapters on orchitis, bulbo-urethral adenitis, and inflammation of the seminal vesicles have been added, and most of the other subject-matter has been brought up to date. The bulk of the volume consists of historical matter, and this is what lends it the only value it possesses. The other subjects have been treated more impartially and less theoretically in other recent publications.

A. P. C. A.

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UNDER THE CHARGE OF

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Green, Blue, Magenta, and Other Colored Urines.—WM. MURRELL (*Edin. Med. Jour.*, 1906, xix, 497) gives a rather extensive report of the various colored urines due to drugs or other causes, which may be of especial interest in relation to the modern synthetic therapeutic compounds.

Blue or Green Urines. A great majority of these are due to methylene blue. He found that $\frac{1}{4}$ grain twice a day imparts a pea-green color to the urine, and when repeated more often, the color becomes blue. These urines decompose slowly and the color which disappears may return on shaking, owing to the absorption of oxygen. All variations between the color of normal urine and that of deep blue may be seen. Green urines may at times be due to biliverdin and occasionally there are indigo-blue urines. The various tests and spectroscopic examinations are then given in detail for purposes of differentiation.

Black Urines. Carboluria: This may be due to carbolic acid or at times to salicylic acid in large doses. The urine is usually dark and smoky. Fuchsin in small doses may produce a dark olive-green urine, approaching black in color. *Melanuria:* This may be due to melaniasis, when there is present somewhere in the body a melanotic sarcoma. Other dark-colored urines may be due to hemoglobinuria and hematoporphyrinuria. The urine is usually of a dark port-wine color. Spectroscopic examination is here most important in distinguishing the cause.

Red and Purple Urines. Aside from the febrile urines which may give these colors to the urine there are certain drugs causing them, such as "Purgen" (familiarly known as phenolphthalein) and largely advertised as a purgative. When the urine is acid there is no color, but when alkaline or neutral it shows a deep crimson-red color. This color is destroyed by acids and restored by alkali. The spectrum is quite characteristic. "Purgatin" is another synthetic drug that colors the urine a bright red in acid or alkaline urines. Pyoktanin, or methyl violet, usually gives a faint violet tinge to the urine. Fuchsin produces a bright-purple urine. It may be readily distinguished from phenolphthalein by the disappearance of its color on the addition of alkalies and the appearance of a yellow color with acids. Murrell gives the various tests for the different drugs and shows in plates their spectra as compared with the solar and urinary spectra.

[Under black urines the author might have mentioned alkaptonuria in which, on standing, the urine may become nearly black.—W. S. T.]

Experiments on the Leukolytic Action of the Blood Serum in Cases of Leukemia Treated with the X-rays and the Injection of Human Leukolytic Serum in a Case of Leukemia.—J. A. CAPPS and J. F. SMITH (*Jour. Exp. Med.*, 1907, ix, 51). The general conclusions which Capps and Smith give in this excellent piece of experimental work are as follows: "The *x*-rays produce in leukemia a disintegration of the leukocytes, affecting especially the young forms, namely, the myelocytes and the nongranular mononuclear cells. A similar action but one of less degree takes place in leukemic blood exposed to the *x*-rays in vitro. The serum of a leukemic patient who has improved under the *x*-ray treatment when injected into animals causes leukopenia; when added in a hanging drop to the leukocytes of another individual, it disintegrates the cells. This leukolytic action is selective, destroying first the mononuclear cells. The strength of the leukolytic action seems to be proportional to the degree of clinical improvement of the patient under the *x*-ray treatment. The serum in a case of leukemia which has been exposed to the *x*-rays has a marked agglutinating action on normal red corpuscles and on other corpuscles. The degree of agglutination varies roughly with the degree of leukolysis. The *x*-ray treatment of normal or leukemic blood in vitro does not materially alter the phagocytic power of the leukocytes. The injection of a strong leukolytic serum from a patient suffering from lymphatic leukemia under the *x*-ray treatment, into another individual with lymphatic leukemia but not under treatment, causes a decided and rapid fall in the number of the leukocytes, the mononuclears being principally affected. With repeated injections, a partial immunity to the serum was established." The nature of the leukolysin remains in doubt. It may be an amboceptor-complement complex or a true toxin.

Review of 105 Reported Cases of Acute Pancreatitis with Special Reference to Etiology; with a Report of 2 Cases. A. EGDAHL (*Johns Hopkins Hosp. Bull.*, 1907, xviii, 130) gives a most careful review of the reported cases of acute pancreatitis with special reference to the etiology, and in summing up notes that the onset was sudden in most of the cases, due to whatever causes, and corresponds closely to the original description of Fitz. As regards the mortality: Of the 44 cases associated with gallstones, 30 died. Of the 32 associated with gastro-intestinal disturbance, 17 were fatal. Of the remaining cases due to miscellaneous causes, 29 in all, 12 died. Sugar was present in the urine of 6 cases;

3 recovered; 2 died. In 1 there was no statement as to the results. Fat necrosis was present in nearly all of the cases operated on or seen at autopsy. As reviewed from the articles in the literature Egdahl notes that the pain is most probably due to the stretching of the sensitive parietal peritoneum, especially where there is much extravasation of blood, and this spreading may account for the different localizations of the pain. The inflammatory process does not necessarily follow the same route as the hemorrhage. The cause of the marked collapse in death has recently been shown to be most probably due to the absorbed trypsin. Egdahl reports on 2 cases of which he has the notes, 1 of acute hemorrhagic pancreatitis in which there was marked inflammation about the pyloric end of the stomach and duodenum, with some occlusion of the ampulla of Vater. No gallstones were present at autopsy, but may have been previously. Fat necrosis were present. The second case was one of acute suppurative pancreatitis in which typhoid fever was most probably the cause. In this latter case sugar was present in the urine and fat in the stools. The head and body of the pancreas were much destroyed. There were no gallstones present.

His conclusions are as follows: (1) Gallstones are probably the most common single cause of pancreatitis. Judging from this series about 42 per cent. of the cases of acute pancreatitis are associated with gallstones. (2) Gastro-intestinal disorders are the next most common cause of acute pancreatitis. In this series of cases about 30 per cent. belong to this group; 17 out of the 32 of this group gave a history of alcoholism. It is very propable that retrojection of bile may be caused by intestinal disorders closing the papilla of Vater. (3) Acute pancreatitis may be caused by many other conditions. Among these we have typhoid fever, tuberculosis, mumps, trauma, syphillis, emboli, appendicitis, malaria, and gastric ulcer. (4) The organism most commonly present is B. coli communis; next comes the streptococcus and the staphylococci. (5) Typhoid fever is the most common of the acute febrile diseases present according to the previous histories of these cases of acute pancreatitis. In the present series 3 were directly associated with typhoid fever alone and 4 had suffered from typhoid fever some time before the onset of acute pancreatitis and had apparently recovered; another had had typhoid fever and erysipelas. But 2 out of these 8 cases were associated with gallstones. It should be kept in mind that in this latitude typhoid fever is the most common continued febrile disease.

The Treatment of Tuberculosis by Marine Climates.—Wm. Ewart (*Medical Press and Circular*, London, 1907, lxxxiii and lxxxiv, 447 and 476) begins this thorough analysis of the effects of marine climates by the following summary of cases suitable for such treatment: (1) All cases of surgical tuberculosis and scrofula are suitable. (2) All latent or threatened cases are suitable.. (3) Early cases of declared tuberculosis are debatable. They are all fit to try the mountain cure, but failing in this not all will benefit most at the sea-side; and more equable climates may be indicated. (4) The suitability of cases of advanced phthisis can only be decided in relation to the existing prognosis and to personal toleration and preference in regard to climate. (5) The excellent progress maintained at chosen spots along the coast by some cases of chronic quiescent phthisis reveals the fact that there are temperaments for which sea-side air is especially favorable, and may be of positive advantage even at the later stages. Ewart goes carefully into the different aspects and effects of sea-air and sea-water, also the different qualities of pure ocean air and sea-coast climates in tuberculosis, and in summing up, his conclusions are that in most cases the inland and altitude cures are more beneficial, but in certain other cases the marine climate is the best, and this latter is of the greatest benefit in the prevention of the disease in suitable subjects. He says that in England especially the best place for the "prevention sanitarium" or "preventorium" is the sea-coast.

Tertiary Syphilitic Fever and the Visceral and other Changes Connected with It.—PARKES WEBER (*Lancet*, 1907, i, 728) reports extensively several cases bearing on the above points, in the first one of which a female on four different occasions showed a remarkably rapid disappearance of the fever on the use of antisyphilitic treatment. The fever is usually of a hectic type and in many cases may often be mistaken for some septic condition or even pulmonary tuberculosis, especially as the fever is sometimes accompanied by chills, night-sweats, and emaciation. Many examples of such cases are reported from the literature. As noted in the accompanying article the diagnosis of typhoid is sometimes made and especially when associated with diarrhœa. Endocarditis may at times be suspected. The associated liver conditions, with pain, tenderness, and even jaundice, may lead at times to a suspected abscess, cholecystitis, etc., and operation may often be tried. Gastrointestinal symptoms in late tertiary syphilis may be very severe and associated with fever, cachexia, anorexia, and obstinate diarrhœa. Obscure bone and joint conditions may often be due to a luetic infection, and when associated with some pyrexia may often be misleading, but clearing up on active antiluetic treatment. Weber gives numerous reports from the literature, with cases and types of fever associated with various obscure visceral manifestations of lues.

This article leads up to the review of another similar article entitled "Two Cases of Syphilis Simulating Typhoid Fever," by D. ROLLESTON (*Med. Press and Circ.*, London, 1907, lxxxiii, 307), who says that the possibility of syphilis being mistaken for typhoid fever has been recognized since Fournier in 1873 emphasized the similarity of the two conditions and introduced the term "typhose" to designate the condition in certain cases of syphilis, particularly in women, which closely simulate typhoid fever. He also gives a brief *resume* of the literature on this subject and notes that, though rare, some of the cases resemble typhoid, especially those with enlarged spleen, abdominal distention, and others with diarrhœa. An interesting reference is to those cases reported by FUTCHER (*New York Med. Jour.*, 1901, p. 1065). The fever may exist apart from any specific manifestations, and to this manifestation Fournier gives the name "essential syphilitic fever." The disease may be mistaken for tuberculosis, malaria, or influenza. Typhoid fever and syphilis may co-exist and a persistent negative Widal reaction may help in excluding typhoid fever; mercury and iodide may be of service. The cases reported by Rolleston were admitted to the hospital with a diagnosis of typhoid fever, the first with a low grade of fever and with marked cerebral symptoms resulting fatally. Rolleston calls attention to the following points in connection with the first case: (1) The tendency that syphilis has to imitate other diseases, as in this case before admission, the diagnosis being, first, diphtheria; second, brain disease; and lastly, typhoid, for which he was admitted. (2) The early implication of the brain. (3) The galloping character of the disease. (4) The intermittent type of fever. (5) The enlargement of the liver. In the second case the general phenomena and the steady high temperature suggested typhoid fever at first, but in a short time typical luetic manifestations appeared. This patient recovered quite rapidly on active antileutic treatment.

The Diagnostic and Prognostic Value of the Enumeration of the Nuclei of Neutrophilic Leukocytes and their Relative Phagocytic Power.—D. M. Kaplin (*New York Med. Jour.*, 1907, lxxxv, 674). As is well known the nuclear polymorphism of the neutrophilic cells extends all the way from a mononuclear form to a form having two or more parts. Arneth in particular took up the study of the relationship of the number of the nuclei of different cells in various diseases. The simple indented and bent mononuclear is most probably the closest to the myelocyte and the youngest of the polymorphonuclear cells. As noted by Arneth the cells predominating in number are those containing two or three nuclei; next, those with one nucleus; then those with four nuclei, and lastly those with five nuclei. In certain pathological states the number of twos and threes may be diminished and those with one nucleus markedly increased. The normal arrangement of figures as regards the number of nuclei may be graphically shown in a count of 100 cells as follows:

Number of nuclei,	1	2	3	4	5
Number of cells,	24	45	25	5	1

Kaplin has been led by this division to make careful nuclear counts and to determine the relation to prognosis and diagnosis in various diseases. He has noted that in the beginning of a disease there is a rapid rise in the count of the one-nuclear cells which increases as the disease gets worse and remains stationary when the disease is stationary. With the subsidence of the disease the number of mononuclear cells diminishes gradually and the binuclear or polynuclear cells increase. He cites many cases with their nuclear counts which bear out these statements and says: "No matter what the number of white blood corpuscles per cubic millimeter, the nuclei of the neutrophiles apparently behave in a constant and regular manner in infectious processes or in any condition where the organism is below par, so that we have a delicate hemic gauge whereby to distinguish an infectious process in low-par states, when a hyperleukocytosis may be absent." The nuclear counts vary apparently with a reinfection or a relapse during the disease. As far as assigning any function to this mononuclear form, he can only offer hypotheses, such as that the extra-formation of these young forms meets the added demand in infections, or that the disease paralyzes the further development of these cells.

As regards the ability of the different nucleated cells to take up bacteria the author performed some experiments with staphylococci and found definitely that the mononuclear form takes up more bacteria than the others, the next in order being the binuclears and trinuclears, those taking up the least number being those with four or five nuclei. This may be graphically shown as follows:

Number of nuclei,	1	2	3	4	5
Number of cells,	32	30	28	10	0
Number of staphylococci in cells,	160	135	126	18	0
Average to a cell,	10	9	9	3.6	0

Kaplin notes in conclusion that this is only a preliminary communication, and says that much further work is needed on this interesting subject.

The Occurrence of Atypical Phagocytic Cells in the Circulating Blood.—M. W. Rowley (*New York Med. Jour.*, 1907, lxxxv, 674). The recent review of an article by van Nuys, in the *Boston Medical and Surgical Journal*, on the presence of atypical phagocytic cells in the blood, brings up this article for review as indicating the possibility of a more frequent occurrence of these atypical cells if only looked for and noted carefully; and by more careful and extensive reports of many such cases much light may be thrown on clinical hematology. Rowley in one case of tertian malaria and three cases of lymphatic leukemia found these atypical phagocytic cells. They varied much in size and shape, sometimes being round and sometimes irregular in outline and shape, with protroplasmic processes. They may have one or more nuclei, the nuclei containing a fine reticular network, and in the majority of the cells being kidney-shaped. In staining by Wright's method the cells resemble the large lymphocytes, the nuclei staining a reddish purple and the protoplasm various shades of dusky blue. There are usually no distinct granulations. The cells may be seen with red blood corpuscles and even other leukocytes engulfed. As to their nature, they resemble to some extent the elements found in exudates into the serous cavities or in the spleen and elsewhere; that is the so-called "endothelial cells." The article has several photomicrographs of these atypical cells, and their presence and phagocytic properties have been identified by good authorities.

[In pernicious estivo-autumnal malaria the presence in the peripheral blood of enormous macrophages is the rule.—W. S. T.]

Cervical Adenitis with Reference to Mouth Infection.—F. D. Donoghue (*Boston Med. and Surg. Jour.*, 1907, clvi, 392) asserts that the frequency with which purulent conditions of the teeth and mouth are overlooked is most important, as in a great many diseases the beginning of the trouble dates from a mouth infection or pyorrhœa alveolorum, which leads first to the involvement of the cervical glands. In reviewing the literature as to the origin of pulmonary tuberculosis he notes that primary tuberculosis of the cervical glands is quite rare, and in a study of 300 cases of enlarged cervical glands at the Boston Dispensary says that in very few cases only should the source of the infection be overlooked, and in all cases before the glands actually disintegrated treatment of the primary cause in the mouth affected a cure. Donoghue then gives a careful description of the various groups of glands of the neck, the parts they drain, and the disease processes they may easily absorb. His conclusions, based on 300 cases, are as follows:

"Enlarged glands of the neck are not primarily tuberculous, and bear the slightest relation if any, to the general or pulmonary tuberculosis. They are due to a mixed infection of pus-producing bacilli. They will quickly resolve if the source of the infection is removed before the glandular tissue becomes disorganized. The treatment in the later stages, especially, is entirely local and surgical, either opening and draining, or, in advanced cases, complete dissection. General hygienic measures should, of course, accompany it."

SURGERY.

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Traumatic Curved Radius, with Progressive Subluxation of the Ulna.—Derscheid-Delcourt (*Jour. de chir, et annales de la Soc. Belgæ de chir.*, 1907, vii, 235) reports a fracture of the radius consequent upon the violent backward turn of the crank of an automobile. In attempting to start a recalcitrant machine he received all at once a shock which he could not control. Severe symptoms followed and the skiagram showed a fracture of the lower third of the radius. There was a wedge-shaped fragment from the ulnar side of the radius, the apex being toward the radial side and the base toward the ulnar side of the forearm. The handle of the crank being held in the fork between the thumb and the fingers, the shock was transmitted through the radial portion of the carpus to the radius, which was thus caught between the carpus and the humerus. To this action was added, probably, that of traction on the lower end of the radius by the anterior carpal ligament. The kind and degree of force in this accident are peculiar to the automobile. The treatment consisted chiefly in retaining the limb in a plaster bandage longer than the usual time, because of the slowness of consolidation. In about eight weeks massage was begun. The skiagram had shown a slight angulation at the seat of fracture, the lower fragment being pushed outward while the upper was directed toward the interosseous space. There was now observed a progressive subluxation of the carpus from the ulna. As the consolidation of the radius progressed, the skiagram showed that the radius became more and more curved inward and forward, and the ulna more separated from the wrist. The ulna was carried backward, the ulnar portion of the wrist being carried strongly forward. The writer explains the mechanism of the deformation by the presence of the triangular or wedge-shaped fragment on the ulnar side of the radius and the gradual tendency of the chief fragments to approximate each other for the production of callus. This progressive shortening and angulation of the radius led to the disarticulation and deformity of the wrist. There is partial functional impotence, that is, supination is very limited, but all the other movements are perfectly free. The patient is, however, considerably distressed by this loss of function, particularly his inability to use the gun in hunting, of which he is very fond, and by the pain on pressure and deformity. Massage and passive movements have been tried. A brace is being employed to support the whole forearm and wrist, furnished with a strong steel spring to keep the wrist reduced. A metallic plate with a pad over the prominence and elastic bands are used with the hope of finally correcting the curve in the radius. Considerable progress has been made already, although the limb is far from being normal. As a last resort, if the cure remains incomplete the writer thinks it will be advisable to do an oblique osteotomy of the radius, advised by Duplay and given in detail in the recent work of Gyselynck and Lemoine upon the reparative surgery of the hand.

Neuralgia of the Sciatic Nerve Due to the Pressure of a Dermatoleiomyoma. De Beule (*Jour. de chir. et annales de la Soc. Belg. de chir.*, vii, 1907, 239) reports a case in which a woman, thirty-seven years old, suffered for years from a sciatica intense and rebellious to all medical treatment. The neuralgia involved the region supplied by the external popliteal sciatic (peroneal). There were no atrophic troubles; muscular power, circulation and sensation were completely normal, and none of Vallieux's points were detected. On the external surface of the knee above the head of the fibula was discovered a small hypodermic tumor, the size of a cherry. It was without pain to superficial pressure, but on strong pressure there was an attack of the typical neuralgia, although the patient had never suspected that the tumor had anything to do with it. It was excised without injury to the nerve and found to be grooved on its under surface for the passage of the nerve. Except for a slight flattening the nerve was otherwise normal in appearance. Without special reason for doing so De Beule stretched the nerve. The cure was immediate and complete. Histological examination showed the tumor to be a fibroleiomyoma. In several cases it has been shown that these myomas are remarkably sensitive to pressure or become the seats of very troublesome neuralgias, occurring spontaneously from cold or from external irritation. The explanation of this is probably to be found in the contraction of the muscle upon the nerve filaments. The etiology and pathology of these tumors is not known.

The Use of Animal Blood Serum in Surgery. Stuart-Low (*Lancet*, 1907, i, 1221) has been using the old oiled-silk dressing applied directly to recalcitrant wounds. The good results in a number of such cases impressed him very much. In trying to explain the results he came to the conclusion that they must be due to the blood serum (or lymph) and the assistance given to the surface circulation. A dry dressing in contact with a healing wound absorbs the normally exuding serum and becomes adherent to the growing tissues. It thus blocks the exudation and therefore the surface circulation and dries the granulations. The oiled silk does not, and keeps the wound always bathed in serum, thus aiding the superficial circulation in the healing region. This dressing, once in common use in surgery, went out of practice because it cannot stand boiling or immersion in strong antiseptic solutions. The writer simply washes in sterilized or boric solution before applying it to a wound. Being impressed with the theory of opsonins and the practical application of them, he argued that if so much depends on the blood serum, it would be well to increase the amount of it in and about wounds; to employ it as the fluid to sluice, irrigate, and dress wounds. To test this view he obtained a quantity of fresh blood from the butcher, the blood being poured directly from the aorta of sheep into sterilized vessels. This was kept in air-tight glass jars. It was mostly in ear cases that he used the serum. It is particularly suited to these, since there is so much bone excavation that a dry instead of a weeping wound results. Desiccation is usually inimical to the ready healing of a wound, and this is successfully avoided when the serum is employed. In every instance the soft structure healed by first intention.

Clinical Remarks on the Operative Treatment of Fractures.—Lane (*Brit. Med. Jour.*, May 4, 1907) says that while recent fractures of the femur can usually be readily and effectually dealt with by operation practically without risk, the surgical treatment of malunited fractures of this bone is often difficult in the extreme. It occasionally causes some anxiety because of the profuse hemorrhage which may take place during the operation. In oblique ununited fractures Lane generally makes an oblique section of the fragments, rather than to freshen the fractured surfaces with the curette or chisel, or to make a transverse saw-cut which would in most cases sacrifice too much length of the bone. Much care is usually requisite in determining the plane in which each fragment must be divided. The fragments are then splinted together with plate and screws.

Fracture of the epiphyseal line of the head, or separation of the upper epiphysis of the femur, is rarely recognized at the time of the injury. Secure union is effected by an abundant callus with great rapidity. The patient suffers from two disabilities: an excessive outward rotation of the lower limb and an impaired function of the hip-joint. Lane has many times operated on these fractures. The capsule is exposed and the anterior ligament cleared and defined. If the stump of the neck is excessively prominent, as it usually is, enough is taken from it to allow more complete range of flexion. The outward rotation of the shaft is overcome by lacing up the anterior ligament transversely to the length of its fibers by silver wire. This approximates the points of attachment of the ligament until the range of rotation of the limb equals that of its fellow. The results have been to bring the limb nearly to the normal in function and appearance.

Concerning Suture of the Patella and its Technique. HAUSCH (*Zent. f. Chir.*, xxxiv, 1907, 530) says that an increasing number of surgeons are suturing the fragments of a fractured patella soon after the accident. The general rule is to operate if the extension apparatus is seriously damaged, but not if the patella alone is broken. The usual test of the power of extension is the ability or the lack of it to lift the extended limb from the bed. The degree of separation of the fragments has some value. Hausch, however, shows by one case, operated on thirty-six hours after the accident, that complete loss of function may exist with only a slight tear, 1 cm. from the patella, of the lateral ligament of one side. He thought that if operation had been delayed a few days the patient would have been able actively to extend the limb, and that the loss of function in this case was due to the freshness of the fracture, that is, to the pain which prevents contraction of the quadriceps, as well as flexion of the joint. Operation was, however, indicated in this case because the lower fragment was split into several smaller fragments the union of which would probably have been disturbed by the massage and passive movements which would have been practised even if operation had not been done. Hausch agrees with Thiem that primary suture of a fractured patella is indicated not only when the lateral expansions of the quadriceps are torn but whenever a gap can be detected between the fragments, The transverse incision is exposed to the danger of infection of the fracture and the joint through a suture and to pressure on the scar in kneeling. The only objection to the large cured incision above the patella is that the flap may become detached later, but if careful hemostasis is observed, this will not occur. If it is made to consist of fascia and skin it will have a sufficient blood supply. He uses thick silver or albuminium-bronze wire, and at least one of the sutures is passed through the whole thickness of the bone into the joint. If one fragment is too narrow or splintered, the suture should surround the borders of the fragment as was done in the case described in this paper. When there is no splintering of the fragment, the other three sutures are passed obliquely through the bone so that they do not involve the joint. He always sutures over the fracture the periosteum and overlying fascia, and the lateral expansion with silkworm gut. Most surgeons dress the limb in extension and begin passive motion in about eight days, some wait three weeks (Franz Koenig) and even four weeks. Moullin, however, does not immboilize the knee after the suture, beginning as soon as possible with massage and passive motion, and permitting the patient to stand in three weeks. In recent times Hausch has begun massage of the quadriceps seventy-two hours after the operation with careful passive motion; although he has had some anxiety concerning refracture or tearing of the sutures. He has never observed it, however. Von Bruns has shown by skigrams that the sutures frequently tear out, and Hausch thinks this is due to strong tension put upon the sutures by attempts at flexion. In the cases which he has seen many weeks were required to obtain right-angled flexion. For this reason he places the knee at an angle of about 100 or 110 degrees as soon as the sutures have been placed in the fragments and lateral ligments. If they do not tolerate this degree of tension they are then loosened and retightened with the knee in that position, which the sutures will tolerate. The wound is then closed, a compress applied, and a suitable dressing to keep the limb in flexion. On the day of the operation massage is applied to the thigh and leg, and in forty-eight hours gentle passive motion. Extension can be made complete with ease, while flexion can be carried from the primary position about 10 to 20 degrees. Gradually the patient is permitted to employ active movement in the knee. Eight days after the operation the sutures are removed and on the fourteenth day with a supporting bandage he is permitted to stand up. Hausch believes that this not only hastens functional healing, but is a safer method.

An Advantage of Operation in the Acute Stage of Appendicitis.—LILLIENTHAL (*Amer. Jour. of Surg.*, 1907, xxi) considers that operation in the usual "acute appendicitis" is practically as safe as in the interval. In the interval we practically make a well man sick, while the individual suffering from the acute disease has his forces, opsonins, and leukocytes prepared for a more serious battle than that with the surgeon. To such a patient the operation comes as a comparatively little thing, and if no error in technique mars the surgeon's work, relief is immediate and convalescence speedy. The operative statistics, always excepting cases of perforation, thrombosis, gangrene, etc., are quite as good as in the interval operation; and if perforation, thrombosis, or gangrene is present, it simply means that operation was all the more indicated.

Report of a Dislocation of the Upper End of the Fibula.—GOLLEY (*Amer. Jour. of Surg.*, xxi, 1907, 171) says that there have been about 25 recorded cases of dislocation of the upper end of the fibula. He reports a case in which a man, weighing one hundred and eighty pounds, slipped and, after sliding, sprawling, and twisting, finally landed in a sitting position with his left leg under him. At the same time he felt something snap. On the following day by flexing and extending the foot a grating sensation was felt in the calf of the leg. By palpation an outward and forward dislocation of the upper end of the fibula was discovered, which became displaced as much as or more than the width of the bone whenever the foot was flexed or extended. A plaster cast was applied and left in place for three weeks, when the dislocation was found cured.

The Pathology and Treatment of Appendicitis.—ALBU (*Mitteilungen a. d. Grenzgeb. d. Med. u. Chir.*, 1907, xvii, 349) says that all cases of appendicitis, in which signs of peritoneal irritation are present, severe or suspicious, should be operated on as soon as possible. The time of operation will be decided by the occurrence of the first perceptible warning symptom of the threatening peritonitis. There is a large number of acute cases of appendicitis, which in a few days will subside spontaneously and be cured, in many cases permanently. There is nothing to justify a fundamental rule for early operation in all cases of acute inflammation of the appendix. In mild cases the dangers of the disease are not greater than those of operation. Early operation in the first twenty-four or forty-eight hours must naturally lead to many errors in diagnosis, since the symptoms at this time, especially in mild cases, are not well developed.

The statistics of early operation have no decisive value, since they include many errors in diagnosis and also the mildest cases. The presence of an appendicitis can be accepted only when the appendix shows macroscopical changes. In the treatment of perityphlitis, even of the mild cases, the use of opium and purgatives are to be avoided. In chronic intermittent and remittent perityphlitis the operation is always indicated, and the earlier the more severe the attack or the symptoms complained of. In lingering chronic appendicitis the operation is to be advised if the objective symptoms (constant resistance and tenderness on pressure at the typical place) are present. When, however, the subjective symptoms only are present, a longer trial should be given to internal therapy, which in such mild cases often leads to permanent healing.

Investigations Concerning the Etiology of Appendicitis.—Kretz (*Mitteilungen a. d. Grenzgeb. d. Med. u. Chir.*, 1907, xvii, 1) says that in November, 1900, he had the opportunity of doing postmortems on three cases of appendicitis one after the other. One of them died one year after the attack, from traumatic tetanus. The others were severe acute fatal cases, terminating in a phlegmonous condition of the appendix and septic peritonitis. Adrian believes that there is an intimate causal relation between tonsillitis and appendicitis. Kretz supports this view on the basis of his observations. Adrian considers the tonsil as the primary focus which gives rise to a bacteremia and that to an appendicitis. Of 3579 postmortems at the Kaiser Franz-Joseph Hospital, 53 were in cases of appendicitis. Of the 53, 19 showed signs of recent acute phlegmonous inflammation. Of these 19, in 14 a complete anatomical examination was made, while in 5, for certain reasons, the neck was left alone. The 14 which were completely examined showed, besides the fatal appendicitis with septic peritonitis, a tonsillar angina. Bacteriological examinations showed, in 12 cases, streptococci in the tonsil and intestine and peritoneum. The changes in the tonsil in these cases are marked, uniform, and typically inflammatory. Kretz's investigations lead to the conclusion that phlegmonous appendicitis can be considered anatomically as a disease resulting from tonsillar angina. He feels convinced that the dominating importance of the pyogenic infections associated with anginas is very imperfectly appreciated. In 1903 he saw 11 such cases without wounds, 15 puerperal processes, and 25 erysipelas cases; not less than 14 acute and 23 subacute pyogenic infections, which took origin from the lymphatics of the throat. The acute fatal cases were 3 phlegmonous anginas, 2 tonsillar pyemias, 1 purulent basillar meningitis, 4 phlegmonous appendicitis cases, 2 of osteomyelitis, and 2 of ulcerative endocarditis. The 23 subacute consisted of 8 cases of endocarditis, 4 pleural empyemas, 2 of osteomyelitis, and a suppuration of the gall-bladder from gallstones. These important and frequent diseases, on account of their various anatomical forms, are not considered by most physicians as being related to each other. They are related to each other, not only as they may be due to one pyogenic microorganism etiologically, but they may belong to the same epidemic group in which the point of contagion is in the angina.

THERAPEUTICS.

UNDER THE CHARGE OF
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Bier's Hyperemia in Acute Gout.—Alkan (*Die Therapie der Gegenwart*, 1907, i, 43), on the ground that blood serum is one of the best uric acid solvents, has applied artificial venous stasis according to Bier's method in the treatment of attacks of gout. This treatment, in connection with local applications, is followed by rapid cessation of the seizure. It is advised to associate alternate local applications of heat and cold with the treatment by stasis. The constriction of the part is kept up for two or three hours, the limb being elevated, and cold compresses are applied, to be followed by hot applications for an equal length of time.

Antisyphilitic Treatment in Diabetes Insipidus.—Vaphiades (*Revue de méd.*, 1907, iii, 296) reports a patient twenty-nine years old, syphilitic for five years, who suddenly became affected with excessive thirst and polyuria; four months later the patient had become markedly cachectic and the urinary conditions persisted in spite of ordinary treatment. At this time the hypodermic administration of mercury protiodide, $\frac{3}{4}$ grain daily, was begun, in connection with the internal administration of potassium iodide in doses of 30 grains daily. After two months of treatment the patient was completely cured.

Gelatin in Melena Neonatorum.—Heyn and Schubert (*La semaine méd.*, 1907, viii, 94) have employed gelatin in intestinal hemorrhages in infants; it is administered subcutaneously, and one patient, an infant of four days, who exhibited icterus and hematemesis, recovered rapidly after two injections, on consecutive days, of 5 drams of a 2 per cent. solution.

The Treatment of Tachycardia.—Hay (*Allgemeine Wiener medizinische Zeitung*, 1907, xiii, 142) considers that treatment can influence the progress toward recovery from tachycardia but little. At times the attacks increase in number as age advances, sometimes they decrease; nevertheless, treatment may lessen the patient's discomfort to a considerable degree. To diminish the number of the attacks we should endeavor to restore the normal cardiac regularity, and to attain this end we should employ general tonic treatment, all mental and physical overexertion should be avoided, and the mode of life should be regulated. All intestinal, uterine, and renal difficulties which may produce reflex excitation should be corrected, and the digestion, in particular, should be regulated. Coffee, tea, tobacco, and alcohol should be forbidden and, during attacks, rest and light diet should be enjoined. In nervous subjects the bromides are useful and the attacks may be shortened by various means. For example, the patient should press his arms and elbows strongly against his chest, compressing the abdomen at the same time, while he holds his breath after a deep inspiration. Good results are obtainable by means of deep, forced respiration, which tends to reëstablish normal heart action. Digitalis is often used, and some consider that it should be given in large doses; it is, however, not always able to give relief. If there is stasis in the portal or general venous circulation, calomel and saline purgatives are indicated. Morphine and applications of ice or sinapisms to the precordium should not be employed unless there is marked cardiac distress. The blood pressure should be carefully watched; during an attack the venous pressure increases and that in the arteries becomes less. Should the arterial pressure be elevated the hippurates, benzoates, and nitrites are indicated. Calcium chloride should be given if the vascular contractility is irregular. As a whole the treatment of this condition is empirical, and we should endeavor to find a drug or combination of drugs which is capable of lessening the excitability of the auriculoventricular band of muscular fibers.

The Treatment of Dropsy by the Deprivation of Salt.—HADFIELD (*The Practitioner*, 1907, vi, 821) considers that there are three theories upon which this form of treatment is based, and which, of the many which have been advanced to explain the efficacy of this therapeutic measure, are worthy of special attention. They are as follows: (*a*) The retention of chloride in kidney disease is due to the morbid condition of the kidney itself, a condition which leads to relative impermeability of the organ to chlorides. (*b*) The second theory maintains that the retention is a simple physical process, entirely secondary to the retention of water in the body. (*c*) The third theory holds that in dropsical patients the body tissues are in some way altered, so that they are enabled to hold, in combination with themselves, a larger amount of salt than usual. These theories are discussed *pro* and *con* and the author states, concerning three patients suffering from ascites and one affected with chronic nephritis, who were treated alternately on saltless and salt containing diet, that in each instance it was found that deprivation of salt seemed to exercise a beneficial effect, not so much in dispersing the fluid as in delaying its accumulation. The results obtained were less successful than have been obtained by this means in France, but the benefit accruing to the patients was quite sufficient to justify the employment of this method of treatment.

Opsonins and the Specific Treatment of Tuberculosis.—SQUIRE (*Brit. Med. Jour.*, 1907, i, 1417) holds that it appears, with our present knowledge, that in those diseases (such as tuberculosis) in which the bacteria produce endotoxins and which are, for a time at least, localized, injections of toxins may be useful in prophylaxis, but are of little use in the actual cure of the disease. He is still unconvinced of the wisdom of attempting to cure pulmonary tuberculosis by the introduction into the body of a further supply of tuberculous toxin. The procedure seems too risky with our present incomplete knowledge of the formation of antibodies and of the conditions which regulate the specific protective action of the leukocytes. Tuberculin may be a valuable adjunct in the treatment of surgical tuberculosis and possibly in tuberculosis of internal organs other than the lungs, but it is inapplicable in pulmonary tuberculosis. Three patients are cited in whom tuberculin treatment administered with all the precautions elaborated by Wright appeared to light up quiescent mischief in the lungs. In conclusion, Squire states that he fears that we have not the cure for consumption in tuberculin, even when regulated by watching the opsonic index, but it seems possible that we are almost within measureable distance of a satisfactory specific treatment for tuberculosis, and that we are indebted to Wright for much of our progress toward this desirable goal.

Tannin in Bronchiectasis.—Marini (*Jour. des Practiciens*, 1907, xiv, 219) finds that tannin and calcium biphosphate exercise a marked vasoconstrictor action upon the pulmonary tissues; they also modify and deodorize the purulent expectoration. He prescribes four powders daily, each containing 4 grains of each drug. If hemoptysis takes place the quantity of the tannin may be increased by one-half. During the treatment the patient should drink only water, for all other liquids, especially alcohol, are contra-indicated.

The Treatment of Asthmatic Attacks.—Von den Velden (*Münch. med. Woch.*, 1907, xiv, 697), on the ground that theobromine sodiosalicylate possesses a dilator action upon the vascular system, has employed this drug in a number of patients affected with bronchial asthma. His results are most encouraging. He has also used the drug in asthmas of the so-called nervous type and in the catarrhal form of the affection. At the inception of the attack he prescribes 15 grains dissolved in water; if relief is not apparent in fifteen minutes a second dose is given; rarely is a third necessary. In most instances the condition is much ameliorated and sometimes the attack is terminated entirely No tolerance to the drug is established and it may be taken daily for a considerable period. Renal and circulatory lesions may contra-indicate its administration.

The Treatment of Exophthalmic Goitre.—Czyhlarz (*Wien. med. Presse*, 1907, xi, 413) considers rest an important factor in the treatment of this condition, especially when emaciation is a marked symptom. Exercise and high altitudes should be avoided and the sea-side does not agree in some instances. Carbonic acid gas baths and chalybeate and carbonic acid waters are often useful. Other hydrothreapeutic measures which sometimes are beneficial are packs and sprinkle douches. An ice-bag may be applied to the enlarged thyroid gland and galvanization of the sympathetic nerve may be undertaken. Iron is especially indicated if chlorosis is present, and the preparations of digitalis may be employed to combat the tachycardia. Quinine and arsenic are sometimes useful as tonics, and sodium phosphate has been recommended by Kocher. Iodine should not be given, but the bromides may be prescribed as symptomatic remedies. Antithyroid medication gives varying results, and the same is true of the administration of thymus extract. With regard to operation it may be said that resection of the sympathetic nerve is useless, but partial thyroidectomy may bring about a cure. Sorgo reports 30 per cent. of recoveries, 51 per cent. of ameliorations, and 13 per cent. of deaths. Radiotherapy has been advocated and has caused improvement in some instances; it is especially potent in bringing about an increase in weight. In conclusion the author states that the most valuable form of treatment is partial thyroidectomy.

PEDIATRICS.

UNDER THE CHARGE OF

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Cerebrospinal Meningitis of the Apoplectic Type, with Hyperpyrexia.—H. F. Warner (*Brit. Med. Jour.*, 1907, i, 992) reports the case of a girl aged ten, who previous to onset had been quite well. While at school she vomited once without apparent cause; later in the day she complained of pain under the left jaw, where there were some enlarged glands, but nothing was thought of it until later, when she suddenly fell from her seat in a convulsion. She remained stuporous from then on until death. Examination showed dilated, equal pupils, retracted head, rigidity of neck and limbs, increased knee-jerks, bilateral Kernig's sign, dissociated eye-movements and divergent strabismus, clenched hands, with occasional bilateral convulsive movements, knees drawn up and other joints flexed. Breathing was stertorous and rapid, pulse 148, temperature 98°. The next day the knee-jerks had disappeared, the Babinski reflex was present, the stupor had deepened, and the temperature rose to 104°. An examination of the eyes showed slight optic neuritis, and a lumbar puncture resulted in the withdrawal of clear fluid, from which was cultivated a large Gram-positive coccus, but no meningococcus. She had two convulsions during the day; in these she became markedly cyanosed, her respirations increased to 85, her temperature rose to 106,° and her pulse could not be counted. She died shortly after the second convulsion. There was no definite rash, but an evanescent, erythematous mottling of the skin could be observed for a short time. The whole illness occupied only 37½ hours. The autopsy showed markedly engorged meninges, but no thickening of membranes or exudate; no gross change could be found in the brain. The spinal cord was surrounded by a clear, gelatinous material. The spleen was enlarged, as were also Peyer's patches and the lymphoid follicles of the intestines; the thymus also was large. There were no signs of tuberculosis anywhere. Warner considers this a case of meningitis, the organism producing it having been of such virulence as to have caused death before the usual exudate had time to form. The optic neuritis also must have been toxic.

Thrush and its Severer Effects.—In the space of ten years J. GORDON SHARP (*British Journal of Children's Diseases,* 1907, iv, 292–296) has seen 7 children dying from thrush as a primary affection. The average age at death was eight and a half weeks, and none was over thirteen weeks. The immediate cause of death in 3 instances was laryngeal implication; in the other 4 progressive wasting, with vomiting and diarrhœa. The fatal cases occurred among people who may be termed "dirty," but the disease occurs in well-to-do circles as well. Families living near ash-pits seem to furnish more cases than those living a distance from them, and improved sanitation has diminished the number of cases in the last few years. Summer diarrhœa is found more frequently among children who have or have had thrush, and Sharp is of the opinion that the Saccharomyces albicans is the cause of it in such instances. Rarely does the fungus grow in the intestines, but the diarrhœa and soreness are due to decomposing food, fungus, and tissue carried down. The treatment of thrush consists in general and local measures. The former consists of castor oil to clear the intestines, a thorough cleansing of the bottles, and dilution of the food to 1 part of milk to 3 of water; whey or weak beef-tea should be given in cases of diarrhœa. The local treatment is attended to by the physician himself. A clean, soft rag is wrapped around the handle of a teaspoon dipped in warm water, and the entire surface is cleansed of fungus and adherent matter until it bleeds. Powdered boric acid is next applied to every part of the mouth; the same procedure is then gone through with the nasopharynx and the epiglottis, if necessary. The nipples and the breasts of the nursing mother must be washed off with clean, warm water twice daily. If the external ear is implicated carbolic soap must precede the boric powder.

A Peculiar Outbreak of Erythema Nodosum.—W. LANGFORD SYMES (*British Journal of Children's Diseases,* 1907, iv, 281–284) reports an epidemic of erythema nodosum, occurring in one of the Homes for Children. The house was large, airy, detached, and had apparently good sanitation; there was very little overcrowding, and there was nothing peculiar in the food of those affected. There were 13 cases in all; 1 of them was followed within twenty-four hours by symptoms of meningitis, and death followed two and a half days after the onset of the erythema; another patient became profoundly ill, temperature going to 104°; 4 developed endocarditis as a part of the infective process; 4 of them had relapses. The outbreak emphasizes the following facts: erythema nodosum may become a serious and dangerous disorder; it is sometimes epidemic; it appears to be of an infective nature; endocarditis may arise during its course; it may be quickly followed by meningitis.

The Medical Treatment of Congenital Pyloric Stenosis.—SUTHERLAND (*Lancet,* March 16, 1907, i) refers to the medical treatment of this affection by means of dieting and gastic lavage. He reports 3 cases which he has treated successfully by such measures. He insists that there will be fewer and fewer cases in which surgical interference will be called for, as our knowledge and experience of the medical treatment advance. The first sign of progress toward cure is cessation of the vomiting. This usually follows quickly after lavage and suitable feeding have been carried out. Then the bowels begin to act naturally. This is a very important sign of progress, for it is in such marked contrast to what occurs in untreated cases. After the medical treatment has been begun the bowels act two or three times a day without artificial stimulation, and the motions are larger and consist of fœcal matter. The stomach peristalsis gradually becomes less marked, and eventually passes off completely. The discomfort and constant whining of the infant also cease. The infant's nutrition improves. This is manifested by a healthier color, increased activity of the limbs, and by a slow gain in weight. These infants cannot be fattened rapidly. Any attempt to secure this will probably end in disaster. A simple diet is best, and the less weighing of the child the better, especially if anxious relatives are about. Diarrhœa is a common complication in the course of treatment. It is due to the fact that the bowel is unused to the presence of food material, and so is easily irritated at first. Diarrhœa is always a serious symptom. Sutherland has not derived much benefit from the administration of antispasmodics, or of opium, even when pushed to their full physiological extent. Saline injections, however, he finds of great service in marasmic babies. From 4 to 10 ounces of normal saline solution can be given daily, and will act as a restorative until the improvement of the pyloric function allows of the entrance of a sufficient amount of fluid into the system by the natural channel. Brandy is also of use as a stimulant. Inunctions of cod-liver oil may be of some value, but in the only case in which he employed it in this way it caused a skin eruption.

Pneumothorax and Acute Hemoptysis in Children.—O. Cozzolino (*La Pediatria*, 1906, 881) described two such cases which occurred in the children's clinic at Genoa. In the first there was a localized tuberculous infection of the tracheobronchial and mesenteric glands in a girl aged twenty-eight months; tuberculous infiltration of the left lung and pneumothorax of the same side. The author gives a tabular analysis of 41 cases: Pneumothorax is very rare in children and is always secondary; tuberculous pneumothorax is, in contradistinction to adults, less frequent than all the other forms of pneumothorax taken together; only about 40 per cent. of cases of pneumothorax in children are tuberculous, the majority of the cases being consecutive to abscess, gangrene, pulmonary infarct, traumatism, etc.

Diagnosis is often difficult owing to either the insidious onset or the absence of important symptoms (amphoric breathing, metallic tinkling, etc.), or to the difficulty of obtaining them in a child that breathes badly. Some of these symptoms, moreover, may be met with in children having pulmonary cavities, pleurisy, or pneumonia.

The cause of infantile tuberculous pneumothorax is either the rupture of a pulmonary cavity by progressive thinning of its walls or the pressure on the lung exerted by masses of swollen, caseous bronchial glands, or more rarely the rupture of an emphysematous vesicle. In Cozzolino's case there was perforation of a large tuberculous cavity in the lower lobe of the left lung, which could not be diagnosticated during life.

Prognosis is absolutely unfavorable, linked as it is with that of the causative disease which contributes to a fatal issue. The most common site for perforation is the base of the left lung. Infantile pneumothorax, whether tuberculous or not, is invariably total and rapidly becomes a pyopneumothorax.

The second case described was a profuse and rapidly fatal hemoptysis in a girl, aged four and a half years, affected with chronic miliary tuberculosis, with bronchopneumonia and a cavity in the left apex. A tabular analysis of 48 cases occurring in children below the age of seven years is given, which demonstrates that profuse and fatal hemoptysis due to tubercle below seven years of age is extremely rare, and occurs only in advanced stages of the disease; there is always a cavity of the lungs, or of the tracheobronchial glands; it is often of a fulminating type, without premonitory signs; its extreme rarity is partly explained by the fact that tuberculosis of the lungs in young children produces hepatization and vascular occlusion rather than destruction of tissue and formation of vomicæ.

OBSTETRICS.

UNDER THE CHARGE OF

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Eighty-eight Cases of Early Tubal Gestation.—Hamilton Bell (*Jour. Obstet. and Gyn. Brit. Empire,* December, 1906) gives the results of his studies in 88 cases of tubal gestation at St. Thomas' Hospital. Only early cases are taken and those under the care of physicians sufficiently experienced to study them accurately. The series is tabulated and the cases are subjected to analysis. Attention is drawn to special points of interest in the various cases. Bell concludes that the diagnosis of tubal gestation followed by rupture or abortion is difficult. In many cases the patient has been ill for one or two months before coming to the hospital, the condition not having been correctly diagnosticated.

Typical signs and symptoms are, the missing of one or two menstrual periods, sudden acute pain in the lower portion of the abdomen on the right or the left side, with faintness and vomiting. At about the same time vaginal hemorrhage occurs, continuing irregularly, the blood lost being usually less than that of normal menstruation. Clots are often passed, and sometimes a cast of the uterine cavity; there is slight elevation of temperature and difficult micturition. When rupture and internal hemorrhage have taken place there will be characteristic symptoms of bleeding. In the majority of cases the hemorrhage is only sufficient to form a pelvic hematocele.

The signs characteristic of this condition are the pushing forward and to one side of the uterus, the cervix being softened and the body of the womb slightly enlarged. Between the uterus and one side forms an elastic swelling which depresses the cul-de-sac in the roof of the vagina, which varies in consistence and tenderness. In 50 per cent. of the 88 cases a period of amenorrhœa occurred, lasting for five weeks or more. In all of the cases abdominal pain was pronounced. It was acute, with faintness and vomiting, in 21; acute with faintness only in

19; acute with vomiting only in 21; acute with faintness or vomiting in 11, and indefinite abdominal pain in 15. The hemorrhagic discharge from the vagina occurring at some period of the illness and persisting was a very constant and important symptom. It was present in 73 out of the 88 cases. The passage of a decidual cast was a very uncertain and misleading symptom. In only 17 of the 88 cases was there a clear indication of this occurrence. Elevation of temperature was present in but 23, but this is unquestionably too low an estimate for a careful observation, and this symptom was not afforded in many cases. It is of little value in differential diagnosis. Disturbance of the bladder was present in 20 of the 88 cases. These cases show that no reliance can be placed upon the occurrence of a period of sterility preceding the ectopic gestation. As regards age, the accident is most frequent between the twentieth and fortieth years. The physical signs varied with the degree of hemorrhage and the source of the bleeding. The condition of the breasts was extremely misleading, while the presence of a pelvic mass was of more value in diagnosis. Softening of the cervix and enlargement of the body of the uterus often gave information concerning the nature of the pelvic swelling. Experienced observers could detect the persistence of the pelvic mass, which was suggestive. Physical signs alone cannot be exclusively relied upon to make a diagnosis. The clinical phenomena must be thoroughly studied and compared. Especially important is the persistence of irregular bleeding. Abdominal pain in the lower portion is next in value. As far as treatment is concerned, most of these cases were treated by surgical operation as soon as the diagnosis was made. Some were treated by rest only, but these were the milder cases in which a positive diagnosis was not possible. When a pelvic hematocele had formed the wishes of the patient and the circumstances of her life should be carefully considered. In the majority of cases recovery will follow whether the treatment is surgical or expectant. Champneys reports 75 cases, in only 26 of which operation was done. As this was reserved for the most severe cases, it was attended by high mortality. In the whole series 7 died, all of them after section, giving a mortality of 27 per cent. In Bell's series of 88 cases, most of them treated by operation, there were 3 deaths with a mortality of 3.4 per cent.; 73 of these were treated by abdominal section, including all the most severe cases. This makes the mortality rate 4.01 per cent. As regards the convalescence of the patients, it is thought that the number of days spent in the hospital, in the cases operated upon, and those not operated upon, is not an accurate criterion. In the first half of the series, the average convalescing was 35.3 days; in the second half, 29.4 days. In Champneys' series the average hospital convalescing was 39.2 days. The mortality was least in those patients operated upon as soon as possible after admission.

This paper and that of Champneys are tabulated and compared in that form. Champneys' patients were largely treated by the expectant method. His mortality was 9.3 per cent., while Bell's mortality was 3.4 per cent. In Champneys', 45.3 per cent. of the patients recovered without operation; in Bell's, but 10.2 per cent. Champneys practised vaginal section in 20 per cent., while Bell could collect but 6 vaginal sections in his cases. The mortality of abdominal section in Champneys' cases was 26.92 per cent.; in Bell's cases, 4 per cent. This striking disparity would indicate that early operation is by far the safer procedure.

Ectopic Gestation Occurring in India.—STANDAGE, of the India Medical Service (*Jour. Obstet. and Gyn. Brit. Empire*, December, 1906), describes 15 cases of ectopic pregnancy occurring in India; 5 of the 15 were European women; the remainder were natives. The ages of these patients ranged from 43 to 22 years. There was but one primipara. The average number of previous pregnancies among these patients was 3.06 per cent. and among the 14 women who had previously been pregnant an interval of 6.07 years had elapsed. But 3 abortions or miscarriages among these patients were recorded.

As regards the signs of ectopic gestation, all but one patient described had distinctly disordered menstruation. On the average 2.2 months passed. Among the signs of ectopic pregnancy there was no nausea or morning sickness. The presence of milk in the breasts was noticed in 4 cases, and 6 had pelvic pain which became sudden in 11. The effect of the pain was not as pronounced as has been reported by some. In fact, among the natives, acute symptoms were comparatively rare. Even after rupture of the pregnant tube the patient would continue about for some time before yielding to the illness. From twenty days to two months was observed as the interval between the onset of pain and the patient's giving up to the condition. In one case a native woman had hemorrhage sufficiently large to fill the pelvis and a portion of the abdomen with blood clot. For a month after the first symptoms this patient worked as usual, and was finally compelled to seek medical advice because of a swollen abdomen. Upon operation the abdomen was filled with blood clot, and a ruptured sac of five months' pregnancy and fœtus were discovered in the peritoneal cavity.

In every case except one in which rupture of the Fallopian tube was either suspected or present, there was a history of a sudden attack of pain. In native patients this pain was never described as agonizing. There was constipation or painful movements of the bowels, and frequent emptying of the bladder when much blood was brought into the pelvis. One patient came to the hospital principally to be relieved of disturbance of the bladder.

The most constant symptom present in all cases but one was irregular hemorrhage, together with a thick, glutinous, reddish or brown discharge. This begins with the expulsion of the uterine decidua, which is usually thought by patients to be a miscarriage. This oozing of blood continues until the condition is permanently cured by operation; 5 out of 15 patients gave a history of abortion occurring at periods varying from four to eighteen days before admission to the hospital.

The detailed report of these cases illustrates how wide is the variation in symptoms and how obscure many of these cases are. The most significant symptom uniformly found was tenderness of the abdomen. This occurred in 100 per cent. of the cases. A tender pelvic tumor was found in 93.3 per cent.; irregular, more or less abundant hemorrhage was also noted in 93.3 per cent. In the majority of cases there was cessation of normal menstruation, and a sudden attack of pain more or less severe. Disturbance in the function of the bladder, vague pelvic pains, and painful defecation occurred in two-fifths of the cases.

A history of abortion with probably the expulsion of a decidual cast was given in one-third of all cases. Milk was found in the breasts in one-fourth of the cases.

As illustrating the tolerance of the Indian native to ectopic gestation and rupture, collapse occurred in but 21.4 per cent. The remainder of these patients did not even go to bed. In none of the cases was there morning nausea and vomiting. The death rate of the series was 7.1 per cent., the fatalities occurring in cases which had become septic, and in cases in which operation was declined.

The Neutrophiles of the Blood in the Puerperal Period, and the Effect Produced by Antistreptococcic Serum.—Burkhard (*Archiv f. Gynäk.*, 1906, Band lxxx, Heft 3) reviews Arneth's work upon this subject. Early in the puerperal period an increase in leukocytes is observed, especially in the cells having a single nucleus. In a case of fatal postpartum bleeding a great increase in the leukocytes to 45,000 was observed: in a case which recovered the number was 15,000. Twenty-four hours later it had fallen to 6000. In pregnancy complicated by nephritis and by lesions of the heart the neutrophiles are not changed. In septic infection there is increased leukocytosis, as a rule, but not as typically and as pronounced as in pneumonia. In puerperal sepsis the number may vary from 2400 to 28,000. In fatal septic cases 2600 to 3500 with 70 per cent. of single nucleus young cells, has been observed. In severe toxemia as few as 2400; and in septic cases, which recovered, 4000 to 5000 have been observed. In fatal pyemia 1800 to 7500 leukocytes are recorded. Other cases with polyleukocytosis ranged from 12,000 to 28,000; polyleukocytosis is an important and significant symptom. In 11 cases of severe infection with streptococci, the number ranged from 6000 to 9000; in 4 cases in which saprophytes, or the bacterium of the colon were present, the number was 18,000 to 28,000. When the septic infection fails to become localized the number of leukocytes is least; when localization becomes established in streptococcic infection, the leukocytosis rises to 12,000 to 15,000; in cases not of streptococcic infection it may rise to 24,000. When improvement begins the leukocytosis decreases. Paultauf's antistreptococcic serum produced improvement in 60 per cent. of the streptococcic cases in which it was used. The serum increased the leukocytes and neutrophiles.

GYNECOLOGY.

UNDER THE CHARGE OF

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A Special Form of Climacteric Hemorrhage.—H. Meyer-Ruegg (*Zentralbl. f. Gyn.*, 1907, xxxi, 629–632) remarks on the greater frequency with which women, flooding irregularly about the time of the menopause, consult physicians. He reports the cases of 4 women, all of whom were passing through the climacteric period, and who came to him because of prolonged serosanguinolent discharge; in several of them it had lasted continuously for weeks. In all of them he found the following conditions: The menstruation had always been profuse and had lasted long; because of this and the profuse leucorrhœal discharge complained of, a preceding state of endometritis could be believed to have existed. The examination showed a smooth, atrophic vaginal mucous membrane; the vaginal part of the cervix was small or entirely absent; the supravaginal part relatively large and the corpus uteri small, hard, and partly involuted. The cervical os was very small and in 2 cases it could only be distinguished because of a thread of mucus hanging from it. After some difficulty a sound could be introduced and the cervical canal was found ballooned out. Dilatation of the cervix brought about an escape of loose, black coagula. He considers the condition one of "cervical hematometra" and believes it to be caused by the prolonged retention of blood behind the small external os. The removal of the stenosis by dilatation or discission brought about a definite cure in each case.

Unexpected Cures in Inoperable Carcinoma of the Uterus.—Unexpected cures in inoperable carcinoma of the uterus are very rare, as are also cases in which for a prolonged period of time no recurrence is noted. Czerny has only been able to report one such. F. Weindler (*Zentralbl. f. Gyn.*, 1907, xxxi, 632–635) reports the details of 3 such instances, in all of which the patients seemed hopelessly doomed and in which they recovered against all expectation. All are living today, five years without a recurrence having been reported. The procedure in all was thorough curettement, cauterization, and application of concentrated carbolic acid. Weindler emphasizes as necessary conditions for such a cure: the full protective forces of the organism against the proliferation of the tumor cells. These are round-cell infiltration in the stroma, increase in the number of bloodvessels in the neighborhood of growing carcinoma, the destructive power to carcinoma toxin of the mastzellen in the lymph glands, and the thickening of the cervical epithelium. Other favorable moments for a cure by palliative measures are a certain degree of emaciation and thinness of the patient, deep action of the cautery, and a favorable histological structure of the tumor.

Extra-uterine Pregnancy.—Between the years 1878 and 1905 Riedinger (*Wien. med. Presse*, 1907, xlviii, 20, 21, and 22) observed among 28,000 patients 42 individuals with extra-uterine pregnancy and 79 with retro-uterine hematocele, altogether 121 cases. Of the former he operated on 14, with 1 death; of the non-operated cases 3 died. Of the 79 hematocele patients 9 were operated on, also with 1 death, while of the other 70 none died. In spite of the relatively favorable results of the conservative measures Riedinger declares himself, because of the finished state of our modern operative technique, in favor of active interference as soon as a diagnosis is made.

Uterine Inversion Following an Abortion.—F. Preismann (*Zeit. f. Geburt. u. Gyn.*, 1907, lx, 152–160) reports the case of a woman, who had previously given birth to 5 children, and in whom without known cause a miscarriage was noted about the fifth month of her sixth pregnancy. The fœtus came away easily, but the placenta remained behind. The patient bled profusely during the next few weeks, but she did not send for a physician until a month later, when after lifting a heavy trunk the uterus suddenly inverted. The placenta was easily loosened then, but the hemorrhage which followed was so great as to cause the death of the patient. A microscopic examination of the uterus revealed marked degenerative changes in the muscle of the uterus about the point of placental attachment. Preismann believes that the placenta, which in none of the reported cases was expelled before the inversion of the uterus, plays a role in the production of the inversion by pulling on the weakened uterine wall; no special violence is needed to bring it about.

Destructive Vesical Mole.—H. KAUFFMANN (*Zeit. f. Geburt. u. Gyn.*, 1907, lx, 136–152) reports the case of a woman of thirty-eight, who had given birth to 11 children, with all of whom she had considerable difficulty because of her flat pelvis and the large size of the infants' heads. Her menstrual flow had always weakened her, but never so much as since the birth of her last child. Because of a persistant bloody discharge she was examined and a diagnosis of vesical mole made; a few small vesicles came away after manual dilatation of the cervix. Curettement was easy and followed by immediate improvement; the patient recovered apparently very promptly. A recurrence was noted within a month and a second curettement resorted to; it was thought to have been a retention, as only a small amount of mole material could be found. Within a few months the patient developed to the right of the uterus a soft elastic tumor, which was believed to be a chorion epithelioma growing from the uterine tissue, but upon operation it was found to be a vesical mole the size of an orange, which had developed in the veins of the broad ligament. It was connected with the uterine cavity and a polyp growing from it by a small pedicle; otherwise the entire organ was absolutely distinct from the tumor. This new hydatiform growth showed no essential difference from the primary growth, but was limited to the different layers of the veins; it must therefore be looked upon as having arisen from the previously removed uterine mole (being a direct continuation of it), and as it extended beyond the limits of the uterus, it has to be classed with the type known as "destroying vesical moles." On account of the limitation of this mass to the broad ligament, the other structures of the pelvis not having been affected at all by it, the tumor is a further proof of the fact that vesical moles may be followed by benign formations in the pathological conception. Such tumors may become malignant at any time, but do not usually become malignant until some new irritant, such as pregnancy, is added. Four months have elapsed since this last operation, but the author is not sanguine as to the definite recovery of his patient.

Chorion Epithelioma Developing Between the Layers of the Broad Ligament.—A. GARKISCH (*Zeit. f. Geburt. u. Gyn.* 1907, lx, 115) reports from Franque's clinic the case of a woman of thirty-seven, who had previously given birth to two healthy children. Within two months she had repeatedly had profuse bloody discharge from the genitalia and was admitted to the clinic because of its continuation. The examination revealed a hard, nodular tumor rising from the left side of the pelvis and another on the right side connected therewith. The operation revealed the mass to be a chorion epithelioma of the left parametrium. The patient died twenty-four days after the operation, of pulmonary embolism, metastases to lung, and pulmonary abscess; metastases had also appeared in other parts of the body, a recurrence being noted also in the pelvis. A careful macroscopic and microscopic examination of the mass produced the following facts and led to the deductions recorded. The case was one of chorion epithelioma which had developed between the layers of the broad ligament and without affecting the uterus. It was most likely preceded by a tubal pregnancy at the isthmus. The tumor developed after the rupture of the tube, from the tubal surface nearest the broad ligament; it developed by growing into the layers of the ligament, separating them as it developed. The possibility of an ectopic chorion epithelioma following an intrauterine pregnancy and miscarriage is not denied. The central portion of the Fallopian tubes showed marked decidual change; the uterine and abdominal portions showed none at all. The uterus presented a typical decidua, 1 cm. thick; the surface epithelium of this had markedly flattened and had proliferated into several layers; the glandular epithelium also showed remarkable proliferation. There were decidual changes on the surface of the ovary. The case belongs to the most malignant forms of chorion epithelioma, as the tumor was found extending into the abdomen within a few weeks after the last period and death occurred four months after the last menstruation.

OTOLOGY.

UNDER THE CHARGE OF

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The Operative Treatment of Othematoma.—H. Seligman's (*Archiv. f. Ohrenheilk,* 1907, vol. lxix) operative treatment differs from the ordinary treatment, by means of simple incision and pressure dressing, in that it aims at a radical procedure in excision of the diseased cartilage, with retention of the protective healthy skin. The auricle having been made surgically clean, and anesthetized by subcutaneous injection of a solution of cocaine and suprarenine (solution of cocaine muriate 0.05 to 10.0 + suprarenine 10.0 to 10 solution, minims 2 to 3), an incision is made throughout the length of the swelling on the anterior surface of the auricle, in its long axis, but through the skin only. There is usually but little hemorrhage, and the two skin flaps thus formed are successively seized by the forceps, dissected up from the underlying swelling, and retracted until the contour of the swelling is completely exposed; a small short-bladed scalpel is the best instrument for this dissection and extreme care should be taken to avoid puncturing the underlying sac, as the lack of the smooth resisting surface which it presents, when intact, considerably increases the difficulty of preparing a perfect skin flap. With the skin flaps completely retracted. and held in position by means of blunt hooks, a circular incision is made about the base of the swelling, and the tissues covering the fluid contents of the sac completely excised. With the evacuation of the fluid there then remains only a concavity upon the surface of which the skin flaps may be adjusted and kept in place by the pressure of gauze pads, until healing has ensued, the ultimate result being a smooth skin surface, with a slight linear scar on the anterior face of the auricle, and a preservation of the natural contour.

This method of operation is equally applicable in the perichondritis of the auricle which sometimes occurs as a consequence of the tympanomastoid exenteration. It is important, however, to distinguish between the serous and the suppurative form of this affection: in the latter the flap operation is sometimes ineffective, in the former it is always applicable; the incision through the skin here also should be made over the greatest convexity of the swelling, in a line with its long axis. Of the 11 cases, upon which Seligman has operated by this method, all have been done upon the anterior face of the auricle.

Microscopic examination of the excised outer covering of the othematoma justifies its radical removal and explains the deformity which usually results in unoperated cases; this outer covering is mainly cartilage, isolated from the main body of the cartilaginous plate of the auricle by means of cicatricial tissue, broken up into individual portions, inelastic, disorganizing and functionally ineffective for the preservation of the contour of the auricle. In cases of spontaneous healing the cicatricial retraction of the connective tissue fibers, evident in the covering of the othematoma, by exertion of varying stress in different directions, under shrinking of the connective tissue coincident with resorption of the fluid contents of the swelling, contribute to the irregularities of surface pertaining in a deformity of the auricle which the method of operation above described is calculated to avoid.

Mastoiditis and Thrombophlebitis of the Lateral Sinus.—E. Bardier and E. Meriel (*Annales des maladies de l'oreille et du larynx*, 1906, xxxii, 140). The early diagnosis of thrombophlebitis of the lateral sinus and jugular vein is one of the important problems of otology today, and any experience which will tend to enlighten the profession upon the clinical evidences of definite pathological changes is worth recording. Bardier and Meriel report a very interesting case of parietal thrombus of the sinus which was suspected but not proved until autopsy. The clinical history and the autopsy findings afford an opportunity for important practical deductions regarding the recognition and treatment of this class of cases.

A young woman, aged twenty-one years, with a history of chronic suppurative otitis media involving the right ear, of eight years' duration, was suddenly attacked by severe pain in that ear, with high fever and rapid pulse. Antipyretic treatment did not relieve the general condition and four days later, when she entered the hospital, the temperature had risen to 105° F., the head pain was severe and she had experienced chills and nausea. Pus from the auditory canal gave a pure culture of the pneumococcus. The right sternocleidomastoid muscle was contracted and painful, but the examiner could not determine any induration along the course of the jugular vein. The patient's general appearance was that of a serious infection, probably arising from invasion of the lateral sinus. Mastoidectomy was performed promptly and the antrum cleared of the necrotic material, but the sinus was not exposed, apparently because the operator had not discovered a direct carious track to that vessel. The temperature immediately fell to 99.5° F. and varied but slightly for two days, but on the third arose again to 105°, with chilliness and vomiting. Then began the typical zigzagging temperature curve, from 100° to 106° F., and a second operation was advised. This time the dural covering of the sinus was exposed, after an exploration of the temperosphenoidal lobe for extradural abscess or meningitis, but, inasmuch as the vessel seemed to present a normal color and was compressible, it was not attacked. The septic course continuing, a third exploration was performed three days later but, still, in spite of the clinical evidences of thrombosis, the surgeon could not persuade himself to open or ligate an apparently normal vessel. Two days later, the ninth from the date of entrance, the patient died of general septicemia.

Autopsy disclosed the fact that the lateral sinus and jugular vein, from the torcula to the hyoid bone, was the seat of a parietal coagulation, 3 to 4 mm. in thickness, covered with purulent streaks; about three-quarters of the central lumen of the sinus was permeable. At the base of the right lung three metastatic abscesses were found and there were areas of bronchopneumonia.

Bardier and Meriel draw the salutary conclusion that it would be wiser in such cases to follow the general clinical indications than to rely upon the appearance of the vessel wall. A study of the temperature is of far more value than mere inspection of the vessel or a drawing of blood from its lumen; in this instance, free blood might have been obtained, despite the presence of an extensive thrombus. Success in the treatment of lateral sinus thrombosis depends upon its early recognition and the prompt establishment of a barrier between that thrombus and the heart. When considering the graver sequels of the suppurative otitis media we must remember that a zigzagging fever curve, especially when accompanied by chills, points more strongly to sinus thrombosis than to any other of the possible complications, and, furthermore, when a sinus has been exposed, because the clinical evidence pointed strongly to infective thrombosis, it is more conservative to ligate the jugular and open the sinus, than to leave it alone.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Biology of Diplococcus Intracellularis.—Flexner, (*Jour. Exp. Med.*, 1907, ix, 105) presents a number of new facts in regard to the biology of Diplococcus intracellularis, especially in regard to the autolysis of the organism. The effect of a number of experimental conditions was tried upon the viability and dissolution of this organism outside of the body. Flexner found that the brief vitality of the cultures outside the body is not to be ascribed directly to the exhaustion of the medium or the accumulation of injurious products of growth. Cold favors destruction of the organism on solid media and in salt solution suspensions. But cold is more injurious to the cocci and destroys them more quickly in a weak than in a highly concentrated suspension. 2° C. was the temperature used. When a similar series of concentrations of suspension of the organism was kept at 37° C., it was found that more organisms survived in the weaker than in the stronger suspensions, giving opposite results from those obtained at 2° C.

When Ringer's solution was used instead of normal salt solution, the organism remains alive in suspension much longer. This fact is probably due to the influence of the calcium salt. Normal salt solution is directly poisonous to Diplococcus intracellularis, and it is apparent that the toxic effect of the sodium chloride can be neutralized by the calcium and potassium salts in Ringer's solution.

Disintegration of the organism occurs rapidly in salt solution suspension, and this is apparently due to an intracellular enzyme. It was found that toluol, which kills the cells but which does not prevent enzymic action, hastened disintegration. Suspensions heated to 60° C. undergo rapid disintegration, while heat of 65° or higher reduces the capacity of the diplococcus to undergo disintegration.

Potassium cyanide possesses the power of preventing or diminishing autolysis of the dead diplococci, thus agreeing with the findings of Jacques Loeb, that this substance has a restraining influence upon intracellular enzymotic activity. On removal of the cyanide the diplococci are partially restored to their previous state of autolysis.

This intracellular enzyme, which is apparently liberated when the cell is destroyed and can be obtained by washing the bacteria, was found to have a destructive action on other bacteria besides Diplococcus intracellularis. It is probable that non-Gram staining organisms are more easily disintegrated by the enzyme than are Gram staining organisms. This enzymotic action was not effected by toluol, but when heated to 65° C. it ceased to act on the diplococcus, while 70° C. was necessary to destroy its action for other bacteria.

Diplococcus intracellularis was found to have low and variable pathogenicity. Small guinea-pigs were found to be very susceptible, even more so than mice, to the same dose. After peritoneal injections there is a fall in temperature, and the extent of this fall is larger with virulent organisms and may be taken as an index of the degree of virulence. Within the peritoneal cavity the destruction of the organism is not dependent upon phagocytosis, but can occur in fluid inflammation exudates through self-digestion. Agglutination occurs in the peritoneal cavity. The diplococcus acts upon the guinea-pig by its intracellular toxin, liberated by disintegration of the organism and not by a secreted extracellular toxin. This is shown by the use of autolysates and cultures killed by heat. Autolysates are obtained by suspending masses of the bacteria in salt solution and then filtering or centrifuging until no bacteria remain. This fluid is toxic for small guinea-pigs when injected intraperitoneally. This toxic action is not materially reduced by heating the fluid to 65° C.: when diplococci are heated to 60° C. their toxicity is distinctly reduced.

Among other lesions found in the guinea-pig were small hemorrhages in the peritoneum. As these might bear relation to the hemorrhages in meningitis in man they were investigated and found to be focal. The vessels were not injured or thrombosed, but the endothelium was sometimes destroyed, as may be seen after injections with hemorrhagin of snake venom. These focal hemorrhages follow injections of the autolysate, but more regularly with the centrifuged than with the filtered fluid.

Peritoneal exudate following injection of the diplococcus has in vitro a more destructive action than the blood serum from the same animal.

Absorption from the Peritoneal Cavity.—Buxton (*Jour. Med. Research,* 1907, xvi, 17) continues his studies in absorption. The earlier part of the work, done in conjunction with Torry, appeared in the same journal, 1906, xv, p. 5, when the absorption from the peritoneal cavity and fate of inert particles and typhoid bacilli was discussed. The first part of the present paper deals with an attempt to determine the number of typhoid bacilli present in the blood of rabbits at various intervals after injection into the peritoneal cavity. The blood was found to contain many organisms immediately after large doses were introduced intraperitoneally, but at the end of one hour the number was greatly reduced, and in twenty-four hours practically no organisms were found in the circulating blood, although in some cases a few were found four days after the inoculation. In rabbits that succumbed to the inoculation many more organisms were found in the blood than in those animals which survived four days.

Organisms were found in various organs of rabbits that survived for four days, but they were present in the anterior mediastinal glands in much larger numbers than elsewhere, while in rabbits that succumbed to the inoculations the organs contained a great many more bacilli. In testing the bactericidal power of rabbit's serum, four days after inoculation, Buxton finds that this property is much less than it is in the serum from inoculated rabbits. The serum from normal rabbits varied in its bactericidal power inversely with the square of the dilution, that is, 1 c.c. of serum diluted 1 to 10, kills not one-tenth but, one-hundredth of the number that the undiluted serum kills.

In the seventh part of the whole series of papers Buxton describes the results of his experiments on the effect of minimal doses of bacteria, and discusses the bearing of these experiments upon the question of peritonitis. He finds that bacteria injected into the peritoneal cavity of experimental animals, even in minute doses, reach the circulation in a few minutes, and that this immediate absorption takes place by the lymphatics of the diaphragm.

He, therefore, thinks that his experiments indicate that the semi-sitting position of Fowler following operations for peritonitis is indicated in order to avoid a rush of bacteria to the diaphragm, and that flushing of the peritoneal cavity is contra-indicated. Buxton believes that the sudden death of rabbits following inoculations is due to toxemia, and that the deaths occurring within one or two hours after operations for peritonitis usually attributed to shock, are analogous.

The Changes in the Liver in Eclampsia. Since Jürgens first described "hepatitis hemorrhagica" as a characteristic lesion of the liver in eclampsia, the condition has been repeatedly studied. Schmorl, in 1893, reported a study of 17 cases and in his descriptions distinguished between hemorrhagic and anemic necroses in this condition.

Konstantinowitsch (Ziegler's *Beiträge*, 1907, xl. 483) reports, from Marchard's laboratory, a study of the livers from 30 cases of eclampsia. He finds, as one of the earliest changes, alterations in the liver cells and of the endothelium of the capillaries in the peripheral part of the lobule. The changes, which consist in granulation and vacuolation of the protoplasm, increase as the disease proceeds until the liver cells present a reticulated appearance. One sees in all cases widening of the capillaries with congestion, and frequently escape of blood from the capillaries, in the peripheral part of the lobule. This is due to slowing of the circulation which is brought about directly by primary thrombosis of the capillaries in the periphery of the lobule. Konstantinowitsch lays great stress upon this thrombosis, and believes that the death of the liver cells goes hand in hand with the development and spread of the thrombosis. First the smaller and then the larger interlobular vessels are thrombosed. The thrombi are, for the most part, fibrinous, or composed of blood and fibrin, and are rarely if ever hyaline in character. In the neighborhood of the anemic necroses is found the fibrin which appears first in the periphery of the lobule and seems to precede the necrosis. Liver cells were found in the lumen of the portal and the hepatic veins, but this was considered to be an artefact. Under certain conditions these lesions, which are characteristic of eclampsia, may heal and give rise to a cirrhosis of the liver. In a few instances evidences of regeneration of liver tissue were seen, a process which before this time had not been noted in the lesions in the eclamptic liver.

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ORIGINAL ARTICLES.

GASTROMOTOR INSUFFICIENCY DUE TO PERIGASTRIC AND DUODENAL ADHESIONS.

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Motor insufficiency occurs when there is a loss in equilibrium between requisite labor and power of labor. When the motor power of the stomach is not sufficient to meet the demands made upon it, the viscus will discharge its contents partially only, or it will become entirely empty only after a prolonged period of time.

There are two fundamental causes of gastromotor insufficiency: (1) A deficient muscular tone or atony, which may be called also a myasthenia; and (2) mechanical hindrance to the expulsion of its contents.

Atony of the gastric muscularis is common. The condition may be hereditary. It may occur or follow the severe infectious disorders. It may be present in the anemias and in cachectic and malnutritional conditions. It is present in neurasthenic and psychasthenic and neurotic conditions. Atony without dilatation does not usually present evidences of motor insufficiency of a degree to produce stasis of stomach contents, but dilatation may occur readily with atony under conditions which require a greater power of labor. If too much food is taken at times; if food is eaten too hastily; if there be congenital defect, like gastroptosis; if slight mechanical hindrance be added in the form of adhesions anywhere in the gastro-intestinal tract; if an abnormal tonus of the pylorus occurs from hyperacidity, so often present in neurotic conditions, dilatation may be added to atony.

In the second group with mechanical hindrance, which is usually due to a stenosis of the pylorus, from the many causes of that condition, the power of the gastric muscularis may be normal or even hypertrophic. But with the mechanical hindrance the gastric muscle is incapable of expelling the ingesta, and dilatation and stasis result.

The motor function of the stomach is very important in the digestive process. Motor and secretory power of the stomach are directly related. Normally, acid chyme, passing through the pylorus, excites the secretion of the intestinal and pancreatic juices. It also excites a nervous reflex which closes the pylorus after a certain amount of the acid chyme has passed into the intestine. With lessened acidity of the stomach contents, or with anacidity, it is found that the stomach empties itself much more rapidly than if the chyme were acid. In some instances, if the stomach is loaded with an enormous amount of food, this antacid condition will permit such an enormous quantity of food to pass from the stomach into the intestine in a short time that the intestinal digestion is inadequate to take care of the food, and diarrhœa results. On the other hand, with hyperacid conditions of the stomach, the food may be retained too long, because but a small amount of very acid chyme will be tolerated in the duodenum, and the duodenal reflex keeps the pylorus in a state of spasm. In the latter condition, with atony of the stomach, as in neurotic conditions, there will be a relative insufficiency with or without dilatation.

The symptoms of motor insufficiency depend upon the degree of insufficiency. In atony of the stomach there may be delayed expulsion of the stomach contents, manifested by a sense of epigastric oppression and weight; and the presence of gas or air, with a bloating of the abdomen and eructations of gas. The patient feels frequently as if the stomach were overloaded when but little food has been taken. If the stomach is tested with an Ewald meal, varying degrees of reaction as to the acidity will be found. Upon a full meal of mixed character, and especially if it contain indigestible particles like seed and leaves of vegetables, a remnant of these indigestible things will be found in the stomach after the physiological time of about seven hours, when the stomach should be empty. After a full meal at night the fasting stomach of the morning will be found usually empty.

With severe motor insufficiency due to atony and dilatation, the objective symptoms are oppression and weight after eating, with bloating with gas and belching, burning in the epigastrium, and indefinite pains dependent upon the nervous condition of the individual. In some instances there will be vomiting of food that has remained in the stomach for an abnormal length of time. This vomitus may show decomposition with the presence of organic acids, various bacteria, and varying degrees of normal HCl and the ferments. An Ewald test meal may show normal acidity or may show lessened or increased amounts of HCl and varying degrees of pepsin and rennin digestion. There may also be present lactic or other organic acids; bacteria of various kinds, with yeast cells; and, if there be hyperacidity, sarcinæ may be found. A large meal of mixed character will show at the end of seven hours the presence of food particles, with the reactions and other conditions present after the Ewald meal. Leukocytes are usually present, and are usually undigested. The fasting stomach of the morning, after a heavy meal at night, will usually show in this severe degree of insufficiency the presence of food with more or less normal gastric juice, containing in some instances organic acids, bacteria, and leukocytes. Unless there be some lesion of the stomach, there is no blood, either microscopic or occult.

If there is an atony of the stomach with dilatation, distention with gas will reveal it. This distention does not cause pain, and there are usually no peristaltic waves to be seen.

If the high degree of motor insufficiency is due to mechanical hindrance at the pylorus, the same findings may result with test meals and upon chemical and microscopic examination. If there is present a chronic ulcer, or the ulcer of an advanced neoplasm, visible and occult blood will be found in the stomach contents and in the stools. If there is cancer, the Gram-proof Oppler-Boas bacillus usually will be found. If the chronic ulcer is present, there may be found leukocytes in excess, and some of them digested. If the stomach is distended with air or gas, and if the disease causing the stenosis is an ulcer, either chronic or that due to a neoplasm, or if there exist recent perigastric and duodenal adhesions, the inflation of the stomach will cause pain, with sometimes a board-like contraction of the stomach, from which the patient gets relief by eructations of the gas or by vomiting. In this condition, too, frequent peristaltic waves may be seen passing from left to right through the epigastrium.

If there be a severe degree of insufficiency with stasis, the amount of fluid absorbed into the blood stream is small, and, in consequence, constipation with dry stools, and a diminution of the twenty-four-hour urine excreted result.

Between this high degree of insufficiency and the slight insufficiency which may be called relative, there are varying degrees of the condition which may, however, be recognized if the patient be observed for a long-enough period of time and if the motor power of the stomach is studied by the proper test meals. A study of the motility, with test meals, should always include the Ewald meal, to ascertain the secretory power of the stomach, as well as a large mixed meal, which should be studied not only seven hours after it is taken, but also the condition of the fasting stomach should be ascertained in the morning after a heavy meal the night before.

Perigastric and duodenal adhesions may cause motor insufficiency. These adhesions are most frequently due to cholecystitis. Chronic ulcer of the stomach or the duodenum may also cause adhesions, but ulcer of the stomach is more apt to interfere with its motility by causing pylorospasm or by producing scars which deform the organ. Adhesions due to operations upon the organs involved may also leave adhesions. Adhesions which result from other pathological conditions, like tuberculosis of the peritoneum, typhoid fever, and other ulcerative conditions, are not, as a rule, situated in the region of the stomach and duodenum, and are not, therefore, to be considered in this paper. Perigastric adhesions produce symptoms that may be classed as acute or chronic, depending upon the age of the adhesions.

The patient who has acute adhesions in the region of the gall-bladder, duodenum, and pylorus, suffers, as a rule, from active manifestations of the disease which causes the adhesions. As I have stated, this is most commonly cholecystitis. Therefore, the patient has pain in the epigastrium, which usually centres upon the right side under the costal arch. With this pain there is a circumscribed tenderness upon palpation, and this is very acute if percussion is made upon a small pleximeter, like the point of the finger. When food is taken, pain of varying degree results. Not infrequently vomiting occurs after taking food, with the relief of the pain. If the infection is confined to the gall-bladder, there is no jaundice. If the infection reach the larger ducts, with or without gallstones, there may be jaundice. Associated with the local symptoms there is usually general disturbance in the form of rigor, more or less fever, rapid pulse, with some physical prostration. If the stomach is distended with air or gas the pain is usually severe and is referred to the gall-bladder region, and the tenderness mentioned above is aggravated. Not infrequently the stomach is tonically contracted and is felt board-like under the hand.

If test meals are given, hyperacidity is usually found. It will be found, too, that in many instances the contents of the stomach are delayed in passing from the organ. There is no decomposition, however, of the food remnants, and no visible or occult blood in the stomach contents or in the stools. There is usually no excess of leukocytes.

This condition will continue for an indefinite period, but usually with the subsidence of the local infection in the gall-bladder improvement in the condition occurs, and finally the patient recovers from all of the acute manifestations. Following the acute attack, a relative insufficiency remains. The patient complains of the symptoms already mentioned in relation to simple atony of the stomach without dilatation. Usually relapses occur with new infections of the gall-bladder, and the patient passes through the same series of symptoms. The condition is not infrequently mistaken for ulcer of the stomach or duodenum. There may be some difficulty in differentiating. In chronic ulcer of the stomach near the pylorus, or in the duodenum, there is usually hyperacidity, with a tendency to increased tonus of the pylorus. In some instances pylorospasm may occur, with pain and symptoms so like those enumerated in cholecystitis with adhesions that they could not be differentiated by a consideration of the symptoms alone. With ulcer, however, the stomach tests will usually show, not only hyperacidity, but the presence of occult blood and sometimes visible blood in both stomach contents and the stools. There are also present numerous leukocytes, many of which are digested. If there is much stasis with hyperacidity, sarcinæ are present. The main differentiating point is the presence of blood and the more constant presence of hyperacidity than in cholecystitis with adhesions. Chronic ulcer, like cholecystitis with adhesions, usually has periods of acute manifestations, followed by periods of latency. In the latent ulcer all symptoms may disappear, but usually there is more or less motor insufficiency of the stomach. Chronic ulcer may be followed by mechanical hindrance through the development of scar tissue and stenosis of the pylorus, or by deformity of the stomach, due to scar contraction, with resulting insufficiency of high grade. This is a condition rarely if ever occurring with adhesions, and, consequently, when this stage is reached, there is only the question as to whether the mechanical hindrance is due to scar tissue from ulcer or to neoplasm.

Cholecystitis with adhesions occurs in infective cholecystitis with or without gallstones. It is known that cholecystitis occurs in the course of typhoid fever, and also during convalescence from that disease. The stomach symptoms enumerated above may occur in a cholecystitis from any of these causes, and when they occur the attention of the physician should be directed toward the probability of a cholecystitis. Motor insufficiency due to recent adhesions from cholecystitis requires surgical interference to attack the fundamental disease. Before operation is done, and while the patient is under medical care, the treatment is that usually directed to the cholecystitis. The stomach should be given absolute rest for a time. Hot applications may be made over the epigastrium, and the patient may be kept in good general condition by the use of normal salt solution, given by the rectum, to the amount of 8 to 16 ounces, every four to six hours. Operation for the cholecystitis after the acute manifestations have passed is safer than during the very acute stage.

Old adhesions about the duodenum and pyloric region of the stomach may be the result of former attacks of cholecystitis, occasionally from chronic ulcer, and often, in this day of modern surgery, from surgical operation on the organs in this region. Occasionally these adhesions are in the form of dense, scar-like tissue, which may produce more or less mechanical hindrance by contraction and subsequent narrowing of the gut or of the pylorus, or they may contract, distort, and thus narrow the lumen of these organs. In this event the patient may suffer from insufficient motility, due to dilatation, with atony, or with an hypertrophy of the muscle of the stomach. Here there is usually no pain, but the usual symptoms of insufficiency are present, both objective and subjective. When the stomach is distended with air there is usually no pain, and relatively no tenderness, in the pyloric region. The Ewald and the motor meals may show stasis and decomposition, without any interference with the normal secretions. There is usually no blood and no excess of leukocytes, and no digestion of leukocytes. If the scar tissue is dense and tumorlike, it may be mistaken for the scar tissue of a healed ulcer, or for a neoplasm. But if there is an open ulcer, the presence of visible and occult blood, hyperacidity and sarcinæ in the one and lowered acidity and Oppler-Boas bacilli in the other, will enable one to differentiate. As I have said, these cases are very rare, and when they occur are subjects for surgical interference.

The great majority of chronic perigastric and duodenal adhesions are of the less dense variety, and are important only when present in patients who suffer from an atony or myasthenia of the stomach, due to any of the causes of atony and myasthenia. If such a patient is a neurasthenic or a psychasthenic, or is neurotic from any cause; if he suffers from any of the anemias, or has a severe infectious disease; if he is careless in his eating as to the amount, or if he does not masticate thoroughly; if he overworks, etc., he is then aware of discomfort after eating. As a rule, these manifestations are purely subjective, and are those enumerated in the earlier part of this paper. Many of these patients talk about adhesions, especially those who have had operations. They can feel the adhesions pulling, have a sense of oppression and weight, of burning and of bloating after eating. They have pain in different parts of the abdomen and elsewhere. Many of them suffer from disturbance of the vasomotor system. They may have palpitation of the heart, cold extremities, or a sense of air-hunger; they tire easily, and many of them diet themselves so closely as dangerously to reduce the general nutrition of the body. Many of them submit themselves to further operations. Occasionally the moral effect of a third or fourth operation may result in improvement for an indefinite time; but, as a rule, the secondary operations do not relieve. A careful examination of such patients will show, with the ordinary secretory test meal, varying degrees of acidity, but usually a condition not far from the normal. The normal ferments of the stomach will be found present. With the motor meal the stomach is empty after the physiological time, and the fasting stomach of the morning is always empty and clean after the full dinner of the night before.

There is, in uncomplicated cases, no abnormal thing found in the stomach contents or in the stool.

Such patients are never surgical and are always subject to improvement and cure under the measures which will improve the fundamental cause of the pain and gastric myasthenia or atony. The surgeon has now learned not to operate upon a neurotic subject with gastroptosis. He must not operate upon the neurotic who fancies he suffers because he has adhesions in the belly, the result usually of cholecystitis or of surgical operation. If such patients treated individually are given moral support, a proper mixed diet of good food, have rest with proper hydratic, calisthenic, and other personal management, they will, as a rule, become well.

I do not wish to prolong this paper by the citation of numerous cases which have come under my observation, but I shall relate one which is an example of motor insufficiency due to acute adhesions; the same individual later presented an example of a neurasthenic and neurotic subject, with subjective disturbances of motility, and submitted himself to subsequent operations without relief.

Mr. C. C., aged forty years, a native of the United States, white, presented himself to me on May 1, 1904. He was a travelling salesman, whose early history was not of importance, with the exception that he had had typhoid fever when about twenty years of age, from which he made, apparently, a good recovery. His habits were good. He had always been a large eater, and, as a travelling salesman, had in many instances eaten rapidly, and had often partaken of much pastry at railroad stations. In the fall of 1903 the patient began to have attacks of indigestion, characterized by vomiting, after hasty eating. This was associated with a sense of oppression and some pain preceding the vomiting, and each attack lasted for two or three days. Five months ago he had a severe attack in which there was a sense of distention of the stomach, belching of gas, and a good deal of pain in the epigastrium. There was nausea without vomiting. A physician passed the stomach tube and washed out the stomach and entirely relieved him. A few days after this he had slight jaundice, with discoloration of the sclera, and a continued disturbance of the stomach, with some burning in the epigastrium, oppression after eating, bloating with gas, and belching Since that attack, five months ago, the difficulty has increased, until he is in almost daily discomfort. Usually there is pain, which comes on from one to two hours after eating, associated with a sense of heaviness and with a good deal of gas, which bloats the abdomen, and is with difficulty eructated. There is tenderness over the epigastrium with the pain. He has had no chill and thinks he has had no fever.

The examination at this time shows a fairly nourished man, who states that he has lost about twenty-five pounds in weight during the last six to eight months. An examination of the mouth and throat shows nothing abnormal. The heart and lungs appear normal. The abdomen is moderately enlarged, but symmetrical; the greater curvature of the stomach appears to reach to the umbilicus. There is considerable tenderness in the epigastrium, and especially in the region of the pylorus and gall-bladder. Upon percussion with the point of the finger as the pleximeter in this region, there is marked tenderness. Distention of the stomach, by giving separate solutions of tartaric acid and bicarbonate of sodium, causes considerable distress, and there is increased tenderness with the distention in the region of the gall-bladder. No peristaltic waves are seen, and the patient secures some relief from the distention by belching gas. After five minutes the gas can be felt passing through the pylorus. With an Ewald meal the total acidity is 98, free HCl 58, combined HCl 40, and there is a trace of lactic acid present. Microscopically, there are a few leukocytes undigested, but no blood and no bacteria. Upon a full meal, given at noon, and the stomach tube passed at 7 P.M., small food remnants were found, consisting of meat and vegetable fibers, and starchy granules. The total acidity was 65, of which 42 was free HCl, 23 combined HCl and acid salts, and there was no lactic acid. The microscope revealed no blood, no leukocytes. The patient was given a heavy meal at night, including tea-leaves and some seedy fruit. The stomach was empty upon aspiration, but there was washed out some vegetable tissue, meat fibers, and some of the tea-leaves and seeds of fruit. This wash water was acid in reaction. There was no blood in either the stomach contents or the stools. The blood examination showed 4,240,000 red cells, 82 per cent. hemoglobin, and 12,500 white cells. The differentiation of the whites showed a normal condition. The diagnosis of cholecystitis with motor insufficiency of the stomach was made, and on May 16 the patient was operated upon by Prof. A. D. Bevan. Calculous cholecystitis with adhesions to the pylorus and colon was found. Cholecystectomy was performed. Following the operation the patient had a moderate lower left-lobe pneumonia, from which he made an uninterrupted recovery. On June 15 the patient was well and able to leave the hospital, at which time a motor meal was given and in the physiological time the stomach was found entirely empty.

On March 2, 1905, the patient presented himself again. At this time he stated that following operation he had for a time been in a fair condition of health. Then he became depressed mentally, and at times he felt that he could not live. He stated that his appetite was poor, and he often had a feeling of sourness in the mouth, and belched a good deal of gas. There was no discomfort after eating, but he felt nervous four or five hours after the meal. He had not regained his strength, and when he attempted to follow his occupation he became easily fatigued and had no confidence in himself. In November, 1904, seven months after his operation for cholecystitis, he was again operated upon by a surgeon in a neighboring State. At that time it was stated that his stomach was dilated, and he was told that adhesions were found about the region of the gall-bladder and stomach, and that the appendix was inflamed. The appendix was removed and the adhesions broken up. Ten days after operation he had severe pain under the right costal arch, and a slight wound abscess formed. After recovery from this operation he had a a sense of fulness in the upper right abdomen, and, as he stated, it felt as if it were full of adhesions. He now returned for further examinations. He remained in the hospital for one week, during which time he was given both Ewald and motor meals, with absolutely normal findings. There was no stasis in the stomach, and the secretions were normal, with the exception of moderate hyperacidity. He was advised to take a mixed simple diet of a plentiful amount, to take hot and then cold sponge baths every morning, to rub down thoroughly afterward, to go about his work, but to modify it, and not to do too much, and, above everything else, to try to forget that he had ever had any disease or operation. On June 21, 1905, three months later, the patient again presented himself, complaining of pain in the upper belly, chiefly in the region of the scar of operation. This pain was aggravated by deep pressure, by coughing, and exercise. He required a laxative every day to move the bowels. He had very little discomfort after eating, and that chiefly in the form of a sense of oppression, with the presence of a good deal of gas. A physical examination failed to reveal any evidence of disease. Stomach tests showed normal secretion and normal motility. Again he was advised to follow hygienic methods, and an attempt was made to give him moral support. On January 3, 1907, the patient again presented himself. He reported that he had remained in much the same condition of discomfort as to pain in the belly and a sense of drawing and weight. In September, 1906, he had come to Chicago and had been operated upon by a Chicago surgeon, who stated that he found adhesions about the colon, stomach, and lower surface of the liver, which he attempted to remove. Following this operation he was relieved for a time, but again the pain occurred in the upper belly, with a sense of drawing in the region of the wound. He now complained of pain in the region of the wound, of a sense of contraction and weight, which was worse when he exercised, but was present all of the time. There was very little, if any, discomfort after eating, and this in the sense of some oppression and of some bloating with gas and belching. He did not vomit. The bowels were slightly constipated. Since his operation in February, 1906, he had lost fifteen pounds. He had not worked during the last year. The physical examination at this time revealed absolutely no evidence of perceptible disease excepting the four scars in the right abdomen, three in the region of the gall-bladder and one near the appendix. He remained in the hospital for five days, during which time his temperature was normal, pulse good, the urine normal. After a full dinner, given at 6 P.M., the stomach was found entirely empty at 7.30 the next morning. Upon an Ewald meal the total acidity was 28 without free HCl, 28 combined HCl and acid salts. Microscopic examination showed no blood, a few undigested leukocytes without bacteria. A full dinner, given at noon, showed an empty stomach seven hours later, without the presence even of gastric juice. A stool was normal, and no occult blood was found. The blood examination showed 4,600,000 reds, 91 per cent. hemoglobin, 12,300 whites.

With this condition the patient was advised that he had no disturbance of the stomach which would require surgical interference, but again he insisted upon surgical relief, and in January, 1907, was operated upon by another Chicago surgeon. It was stated that adhesions were found in the region of the old operative wounds and between the liver, stomach, colon, etc. These were broken up, and the patient made a good recovery from the operation. A letter received from him in the latter part of April, conveys the information that he has had no relief from his former sufferings.

This patient presents, as I have stated, examples of motor insufficiency due to acute perigastric adhesions and cholecystitis, for which operation was indicated, and was successful in removing the fundamental cause. But the patient was a neurotic, who continued to suffer from subjective symptoms, and, although subsequent examination showed no evidence of stasis of the stomach due to an insufficiency, he was operated upon three times more, and without relief.

Had the patient been kept under institutional treatment, subsequent to his first operation, for a sufficient length of time to restore his nervous equilibrium, he doubtless would have entirely recovered. In his neurasthenic state there can be no question that there was a general myasthenia, with atony of the stomach; consequently, he suffered from subjective symptoms of disturbed motility, and, above all, he suffered from discomfort incident to the adhesions following four operations, because his nervous apparatus was so irritable that every abnormal thing in his body gave him discomfort.

CHYLOUS ASCITES AND CHYLOUS PLEURISY, IN A CASE OF LYMPHOCYTOMA INVOLVING THE THORACIC DUCT.

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The following case seems worth recording, though incomplete in some respects, on account of the bearing it has upon the question of the relation of leukemic processes to those generally classed as sarcomatous. The true relations cannot be made clear until the exact etiology is known, or at least until the mechanism of the leukocyte mixture is understood. Clinically and anatomically the case would fit into the classification of sarcoma. The picture is almost the same as that in a case reported by M. Herzog[1] some years ago as a lymphosarcoma. But many conditions formerly so classified have been separated on account of the peculiar condition of the blood, or of the minute histology of the new-formed tissue—chloroma, Hodgkin's disease. In this case the blood had a similar change, though the total number of leukocytes was only slightly increased. While it may be said that such cases are merely sarcomas, with accidental cells in the blood, it seems to me more rational to classify the present case as I have.

J. C. G., farmer, aged fifty-nine years, single, American, was admitted to the surgical clinic of the University Hospital, October 1, 1906, on account of an abdominal tumor and ascites, and was transferred to the medical clinic October 8.

His father died at sixty-six years of pneumonia, his mother at eighty of apoplexy. Two brothers are living. One brother died of yellow fever. A sister died of "paralysis." As a child, patient had measles, whooping-cough, varicella, and typhoid fever. Had "fever and ague once." Fifteen years ago had pneumonia; in bed six days. Later, had symptoms of empyema, followed by the formation of a swelling low down in the left side of the thorax. This "broke internally" after lasting a year, and soon after it broke blood and pus were evacuated per rectum. Six years ago a soreness came in the site of the former swelling, tender on pressure, and lasted two years. Four years ago a hard spot came in the midaxillary region, low down in the left thorax. He than noticed enlargement of the abdomen, and a tumor was discovered by Dr. W. F. Hoyt, of Paw Paw, Mich. Dr. Hoyt advised the patient to go to the hospital when he found the tumor was growing rapidly. Dyspnœa became marked, especially on lying down.

On examination in the surgical clinic, December 4, 1902, as I find in the notes kindly furnished me by Professor de Nancrède, "a mass was found extending from under the ribs on the left side nearly to the inguinal canal, and from the right nipple line to the left midaxillary line. The mass was freely movable; it had a blunt edge on the left side, and several notches in the lower edge. The mass seemed to be made up of three or four polyp-like projections." A diagnosis of abdominal tumor was made and the patient returned to his home. Soon after this many glands became enlarged in the neck, groins, and axilla. Some of them afterward became smaller. A year ago the abdomen began to get larger, and finally reached 60 inches around the waist. There was great dyspnœa. There was a grating sensation in the abdomen, audible to others as well as the patient. Under treatment by salts for six weeks, the waist fell to 36 inches. The strength was much reduced by the treatment. The abdomen was aspirated in August and September, 1905, and six and nine quarts, respectively, of pink, opaque, and milky fluid, were withdrawn. Both times large quantities of fluid leaked through the punctures for several days. On admission to the surgical clinic October 1, 1906, six liters of similar fluid were removed by Professor de Nancrède, and several more liters drained through the wound, as before. The patient was then transferred to the medical clinic.

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[1] Lymphosarcoma of the Mesentery. Jour. Amer. Med. Assoc., February 11, 1899, p. 275.

On examination by Dr. F. W. Smithies, October 8, 1906, the following notes were made (abstract): Temperature, 97°; pulse, 76; respiration, 21. Frame medium; muscles small; panniculus thin. Weight 152—about as usual. Patient lies partly on right side, propped up in bed; looks tired; voice weak. Skin cool, dry, inelastic; sallow in general; darker on face, axillæ, and arms. Slight œdema of ankles. Over most of body and extremities are numerous small papular eruptions, and scars of brown, purple, or pink color, in various stages of evolution. Scars of old papular eruptions on face. Cervical glands slightly enlarged, discrete, hard, and painless. In each axillæ are two lymph glands the size of large marbles, rising 1 cm. above the general surface. The inguinal glands are similarly enlarged. Eyes sunken, lids dark, scleræ pale with slight yellow tint; pupils equal; react slowly to light. Cheeks and ears pale; lips pale, slightly cyanotic. Tongue tremulous, pale, thick, marked by teeth; surface dry, with thin coat. All teeth gone. Neck long, thin. Thorax short, shallow, depressed below level of nipples in front and with slight funnel chest. Clavicles prominent. Interspaces retract in inspiration, especially in upper axillary regions. Litten's phenomenon in left from sixth to eighth intercostal space. On the right there is only a faint shadow in the eighth interspace. On percussion the apices are negative. Right lower lung border at fifth rib in the parasternal line. Dulness in the axillæ while lying. On sitting up the line of dulness rises to the fourth rib and extends at that level into the back, where it rises again. On the left, pulmonary resonance to the seventh rib. Slightly diminished below the clavicle. On auscultation, soft bronchovesicular breathing, with piping rales, is heard on right above the dulness; below that sounds almost inaudible. On left, harsh vesicular breathing, especially below the clavicle.

The apex beat is faintly visible and palpable in third interspace just inside of nipple. Upper border of dulness on third rib; left edge in the third interspace just outside of nipple. Right side at left edge of sternum. Sounds faint at apex. The second pulmonary accentuated. Radial artery thickened; pulse small, regular, compressible. Blood pressure, Stanton 12 cm. band, systolic, 100

100 to 104; diastolic, 55 to 62. Veins of arms, thorax, and abdomen dilated.

The abdomen, two inches above the level of the ribs, is roundish, but flattened on top and bulging on sides. Abdomen measures 102 cm. at the navel. Wall tense, resistant, especially in left iliac region. There is a hard nodular mass in the left side, extending from the right of the navel to the left posterior axillary line, and from the margin of the ribs to the left iliac fossa, filling the latter. The upper border runs down from the lower costal cartilages toward the navel. The surface is smooth in general, but with large prominences. The right side of the abdomen is resistant, but deep palpation is impossible. The edge of the liver can be felt on deep inspiration. Spleen not palpable. The mass seems continuous with its dulness in the upper part. There is fluctuation on the right side, less distinct on the left. There is movable dulness in the abdomen, more distinct in the right side.

Urine: Twenty-four hours, quantity 410, and 600 c.c.; specific gravity, 1026; turbid, alkaline (decomposed); no albumin (3 tests); no glucose; no bilirubin; sediment negative.

Blood· Flow rather sluggish; color dark, watery; red blood cells, 4,390,000; leukocytes, 8651; hemoglobin (Miescher), 70 to 80 per cent. For differential counts see below.

On October 9, I found some changes from the condition noted above: "The apex beat is under the nipple in the fourth interspace. The abdominal mass lies more in the middle of the abdomen between the two parasternal lines, and from the margin of the ribs to three inches from Poupart's ligament. It is very superficial, partly hard and nodular, partly soft and elastic. It moves slightly with respiration. The left lumbar space is soft. The lower end of the mass is thick. It gives dulness on percussion. To the right of this mass other nodular masses can be felt an deep palpation."

One of the enlarged axillary glands was removed for examination. The report (Dr. Warthin) is: "Lymphocytoma (lymphosarcoma). Aleukemic stage?"

On tapping the abdomen in Monro's point I obtained a thin, milky, slightly pink fluid, and similar fluid was obtained from the right pleura. The amount taken from the abdomen was 2450 c.c. Specific gravity, 1014. Amphoteric to litmus. Acid to phenolphthalein. It contained 3.5 per cent. of fat. Most of the pleural fluid was lost by accident, and not measured, but enough was left for examination of the sediment (see below).

The patient left the hospital before any further examinations were made. At the patient's home it was not possible to make careful observations. He failed slowly, and died January 1, 1907. Through the efforts of Dr. W. F. Hoyt, I was fortunate enough to obtain an autopsy, which was made by Dr. A. S. Warthin. I give an abstract of his notes:

Autopsy, January 10, 1907, 2.30 P.M.

Clinical diagnosis: Chylous ascites, lymphocytoma. Chylous pleurisy. Abdominal tumor—splenic(?) and lymph glandular.

Pathological diagnosis: Chylous ascites, pleural effusion, lymphocytoma of mesenteric and retroperitoneal glands. Obstruction of thoracic duct by tumor mass continuous with mesenteric tumor. Lymphocytoma infiltration and secondaries in all organs. General atrophy and passive congestion. Lymphoid hyperplasia of bone marrow.

Frame large. Abdomen greatly distended. Through abdominal wall can be felt, in left hypochondrium, a somewhat irregular tumor, extending to about median line at level of umbilicus and a hand's breadth below level of same. In pyloric region a smaller tumor mass can be felt through abdominal wall; somewhat movable, apparently connected with mass on left side. Over this several smaller masses can be felt, freely movable, probably in omentum. Also enlarged glands in neck, more marked in axilla and groin, the largest the size of an English walnut. No tumor of bones or scalp. Lymph glands in groin same consistence as tumor; firm, somewhat elastic, no fluctuation. There is a puncture mark below umbilicus.

Skin pale. Muscles flabby. Rigor mortis present throughout. Panniculus very thin. Œdema marked over lower extremities.

Diaphragm at fourth intercostal space on right; fifth rib on left.

Mediastinum contains large numbers of enlarged lymph glands of somewhat firmer consistency than elsewhere.

Thymus: Large lymphoid masses in thymic fat; on section, uniform medullary consistency, very cellular.

Pleural cavities: Right contains about 1 liter grayish turbid fluid about same appearance as in peritoneal cavity, only thinner. Left, 300 c.c. same.

Pericardium: Sac not distended, fluid slightly increased, clear. Subpericardial fat increased.

Heart: Apex behind upper part of fifth rib, inside nipple line. Somewhat smaller than cadaver's right fist. Contains moderate amount of fluid blood and soft clots. Muscle rather pale, brown, soft; easily torn. Right heart: Auricle contains large antemortem white clots. Ventricle wall measures 2 to 5 mm., 1 to 2 mm. being fat. Left heart: Ventricle contracted. Wall measures 15 mm. Valves: Tricuspid orifice and valves negative. Pulmonary orifice and valves negative. Mitral flaps show nodular thickenings of proximal margin. Chordæ tendinæ thickened. Aortic semilunars show a slight thickening. Antemortem mixed clots extending into coronary artery.

Left lung: Old adhesions at base, free at apex. Slight thickening of pleura. Moderately distended. Moderate anthracosis. Hypostasis in lower lobe. Right lung: Completely collapsed;

cavity filled with grayish turbid exudate. Lung shows complete atelectasis, covered with dense fibrinous exudate. On section lung grayish. Small bronchi dilated.

Bronchi: Smaller ones dilated on left side, mucosa thickened; also on right contained purulent exudate. Bronchial glands: Greatly enlarged, œdematous on both sides; on section, soft medullary consistence; very cellular.

Aorta shows beginning arteriosclerosis.

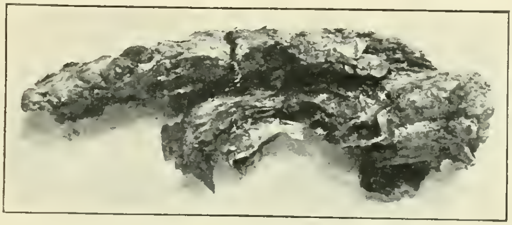

Fig. 1.—Upper part of the thoracic duct.

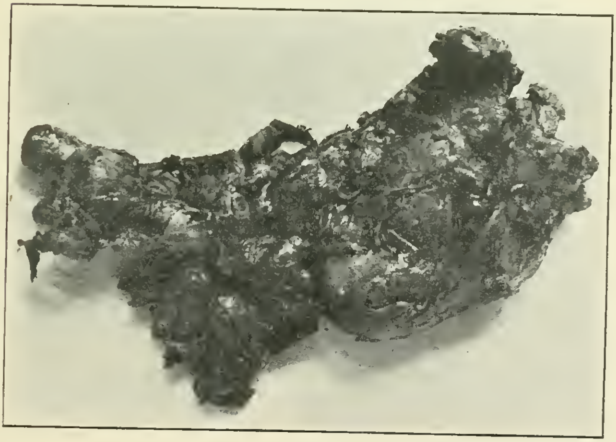

Fig. 2.—Lower part of the thoracic duct.

Thoracic duct: Extending from receptaculum chyli to subclavian is a solid cord of tissue, larger below, where it is about 4 to 6 cm. in diameter, gradually decreasing above to 2 cm., filling up the greatly dilated thoracic duct; on section it is pale, with scattered reddish spots, medullary, uniform consistence; very cellular, and resembling the abdominal tumor and the lymph glands in all respects. Above, this tumor mass projects slightly into subclavian. Below, the large mass in receptaculum chyli is directly connected with the retroperitoneal and mesenteric mass by cords of similar lymphoid tissue. (Figs. 1 and 2.)

Recent peritoneal adhesions around puncture wound. Omentum is slightly rolled up below umbilicus, moderately rich in fat. Contains a number of enlarged lymph glands. On section, these resemble lymphoid masses elsewhere.

Spleen: About normal size, somewhat adherent to diaphragm by easily torn adhesions. Irregular in shape, upper part pale, partly separated from remaining portion on anterior margin by deep fissure running about five-sixths of the way across surface. Bottom of fissure covered by dense hyaline connective tissue, capsule thickened throughout, showing hyaline patches. On section, soft, pulp hyperplastic, covers up stroma to some extent. Follicles diminished in number, a few of larger size found.

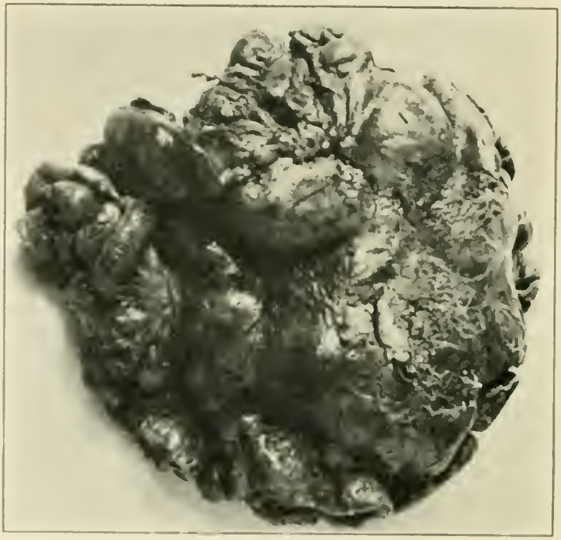

Fig. 3.—Mesenteric growth (with the intestines attached).

Arising from abdomen is a large nodular tumor mass occupying the position of the mesentery and the root of the mesentery (Fig. 3). Entire small intestine borders the lateral aspects of tumor. Smaller nodules and cords extend from main mass up on to intestine beneath serous coat. The mass measures 30 cm. transversely, 25 cm sagitally, 15 cm. anteroposteriorly. Larger part overhangs pelvis, is free, can be lifted up, but at the root of mesentery is continuous with a similar large nodular mass, occupying place of retroperitoneal glands. On section, the mass is found to be made up of nodules more or less confluent, presenting an appearance exactly like that in lymph glands, very cellular, yields abundant exudate, uniformly medullary, grayish white, spotted, and streaked with red.

Adrenals negative.

Left kidney 11 × 4 × 3 cm. Pelvis dilated. Mucous membrane thickened. General appearance somewhat that of right. Over surface whitish nodules about size of pinhead. Right kidney 9×5×2.5 cm. Cortex, 4 to 6 mm. Fibrous capsule strips easily. Bleeds freely, rather uniformly dark-red color; otherwise negative. Outside fatty capsules of both large masses of lymphoid tissue, extending along bloodvessels.

Ureters both dilated, left more than right. Pushed somewhat to left by tumor mass on right side. Bladder moderately distended. Contains about 75 c.c. turbid urine.

Duodenum flattened by pressure of tumor. Wall atrophic; contains thin, yellow, bile-stained material. Common duct also compressed.

Stomach: Walls atrophic, postmortem change. Hypostatic congestion, with small hemorrhage. No enlargement of lymphoid tissue.

Liver 33×20×9 cm. Right lobe enlarged more than left. Capsule thickened around ligaments. Lower edge rounded, fairly firm. On section, moderately rich in blood. Periphery of lobules shows some fat. Consistency rather soft as a whole, central veins dilated. Numerous anemic areas. Lobules large. Islands of Glisson infiltrated.

Gall-bladder filled with thick bile, wall œdematous, serous surface thickened. Contains a number of facetted gallstones; central pigment and coating of cholesterin and lime.

Portal vein compressed by tumor.

Pancreas small, consistency soft.

Portal glands enlarged, same characteristics as elsewhere; vena cava compressed. Retroperitoneal glands enormously enlarged, forming a large nodular tumor, of the same character as that found in mesentery. Section shows soft medullary mass of almost homogeneous consistency, spotted with red. Scraping yields abundant cell juice. Mass extends from diaphragm to pelvis, 30 cm. long, 6 to 10 cm. thick. Prolongations continuous below the tumor masses along external iliac and femoral vessels, extending into pelvis along both sides of bladder.

External genitals markedly œdematous, especially scrotum and tunics of testicle. Body of right testis œdematous, otherwise negative. Left about size of right. Some thickening of epididymis. Prostate about normal size. On section, negative.

Sternum easily fractured; red narrow hyperplastic. Periosteum of sternum (under side) slightly thickened. Can be separated easily; leaves roughened bone surface. Lumbar vertebræ opened in body. Red marrow hyperplastic.

Microscopic findings. The abdominal tumor is made up of cells of lymphocyte type, fine delicate reticulum, no structure of lymph gland remaining, although each nodule appears to represent a lymph gland, remains of a capsule being found in many of them. The capsule is infiltrated with cells of the same type, this infiltration extending throughout fat and connective tissue of mesentery.

Cords of the same atypical lymphoid tissue extend through the lymphatics. The entire mesentery shows more or less infiltration of this character, the largest masses being found near the root. The wall of the small intestine is infiltrated, the infiltration varying in degree in different portions. The solitary follicles and Peyer's patches show similar tumor-like hyperplasia. Mucosa of stomach not involved. The lymphoid growths are very vascular, show numerous hemorrhages, and present very few small areas of degeneration and necrosis. A few mitoses are seen.

The retroperitoneal tumor has the same character as the mesenteric growths.

The mass in the thoracic duct is a lymphoid tumor of the same character as that in mesentery.

Peripheral lymph glands: All show a lymphoid hyperplasia with loss of characteristic lymph-gland structure, a uniform mass of lymphoid cells infiltrating capsules and tissue outside. The distinction between cortex and medullary portion is entirely lost, the sinuses are obliterated; there are no germinal centres. No giant cells or endothelial cells, a few eosinophiles.

Lung: Large lymphomatous nodules replace the peribronchial lymph nodes, and throughout the lung tissue there are nodules of varying sizes similar to that found in mesentery and elsewhere. The large, white, flattened areas of thickening on pleura are found on section to consist of subpleural lymphoid areas, corresponding to those found elsewhere.

Heart: Atrophy; fatty degeneration. No lymphoid areas or infiltration.

Spleen: Atrophy, congestion; the follicles are replaced by masses of lymphoid tissue, corresponding to that found elsewhere, but differing from normal Malpighian follicles.

Kidney: Cloudy swelling, atrophy, small areas of lymphoid infiltration into one kidney large enough to be seen with naked eye.

Liver: Marked periportal lymphoid infiltration, congestion, atrophy, some fatty infiltration.

Other organs: Show atrophy, congestion, some parenchymatous degeneration.

Bone-marrow: Lymphoid hyperplasia of bone-marrow.

So far the case seems not very different from one of multiple sarcoma, or malignant lymphoma or lymphosarcoma of writers, but the condition of the blood, when examined in detail, shows certain points of interest.

For microscopic examination of the blood cover-glass preparations were stained with Wright's stain and examined by counting continuously with a mechanical stage. The red cells show moderate variation in sizes. There are a few cells not more than 3 μ in diameter, a few slightly larger than normal, a few pear-shaped forms. A few very pale cells. No nucleated reds. The leukocytes are chiefly mononuclear, the differential count being (652 cells): lymphocytes, 69.8 per cent.; large mononuclear and transition 0.7 per cent.; polynuclear, 27.4 per cent.; eosinophile, 1.3 per cent.; basophile, 3 per cent. The lymphocytes cannot easily be put into two groups of large and small. Normal small lymphocytes are few—about 1 per cent.—but there are variations in size from cells about the size of a red corpuscle up to cells twice that size, or, rarely, larger. Many have distinct, round but more or less fragmented nuclei, and a pale spongy or reticulated protoplasm in a ring or crescent about the nucleus. Others have paler, often thready or foamy nuclei, and in some no distinction can be made between nucleus and protoplasm, the cell taking the dye faintly, in a fine network, though with its outline is well preserved. There are also cells with notched, incised, reniform, or saddle-bag nuclei, having the general characters of the others in this group. In other words, the lymphocytes show distinct leukemic features. The cells called large mononuclear are few, with large bodies, pale nuclei, round, oval or irregular and very pale protoplasm. The polynuclears show considerable variation in size, up to twice the normal, with nuclei usually large in proportion. There are no very small ones. Their granules stain well and they show only slight tendency to complete separation of the parts of the nuclei. The eosinophile cells are all polymorphonuclear, and of normal size. The mast cells are of medium size, with polymorphonuclei.

I have counted only well-preserved cells. There are many degenerates—45 to 50 of all others. They are all, apparently, of lymphocyte origin and are easily distinguished from the others. Even without these it is obvious the blood is lymphocythemic, though the total number of cells, even with degenerates, is not much increased.

The sediment of the ascitic and pleural fluids shows lymphocytes almost exclusively. There are in both a few larger cells, of endothelial origin, but so few, and so irregularly scattered, that a comparative count could only be made by including many thousands of all cells. The lymphocytes in these fluids show the same characteristics as those in the blood. The endothelial cells are small, as a rule, have few vacuoles or other degenerative features, have deeply-staining nuclei and rather slightly basophile protoplasm.

A CONTRIBUTION TO THE STUDY OF LONG-CONTINUED FEVERS.[1]

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Through the kindness of Dr. F. H. Humphris, of Honolulu, there was referred to me in July, 1906, a lady of forty-seven years with the following interesting history: There had been no tuberculosis or malignant disease in the family. She had been twice married—the first time at sixteen—and had one child living and well. As a girl, she was said to have had malaria. In 1893 she had been infected with syphilis, which was treated three months at Arkansas Hot Springs, and again for some weeks in 1900. For ten years there had been a cough each winter lasting three or four months, annoying rather than serious, and only occasionally keeping her in bed. During these years there had been four or five attacks of so-called "la grippe," attended with fever for a few days, cough, and aching. In 1895 a curettage was done for some slight uterine derangement, but there had never been serious pelvic symptoms. In 1898 an acute attack of abdominal pain was referred to the appendix, and Dr. Murphy, of Chicago, operated later in the year and removed an appendix showing slight pathological changes.

During 1902 and the first half of 1903 she was very well and travelled extensively. In the latter part of 1903 and the first months of 1904 she had three attacks of sudden, severe abdominal pain. The pain was general throughout the abdomen, colicky, lasted a few hours, and was thought due to eating fruit and drinking mineral water. In Paris, during the summer of 1904, she was tired, nervous, and had little appetite, but felt well during the autumn in St. Louis. A little later, in Chicago, there was fever for a few days, with headache and aching in the muscles, and she felt badly for a month. The usual bronchitis kept her rather miserable during the winter of 1904 and 1905 in Paris, and an attack called "la grippe" confined her to the house for a few days in March. In the summer months, in New York, she felt tired, had very little appetite, and was said to have liver trouble. She was much better a little later in Canada and the Northwest, and felt perfectly well when she sailed for Honolulu in September, 1905. The patient had always been a great traveller. She spent 1902 in France, Italy, and Austria. In 1903 she was in Japan, Honolulu, Mexico, France, and Spain; in 1904 in Italy, the Riviera, Greece, Turkey, the Mediterranean and Egypt.

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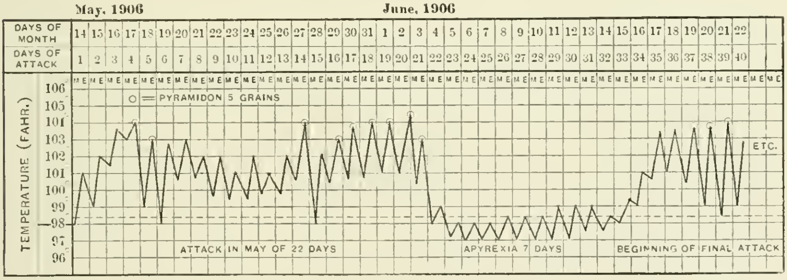
[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 7, 8, and 9, 1907.

The present illness began on the steamer two days before reaching Honolulu, in September, 1905. There was intense muscular soreness, considerable pain in the joints, and fever lasting a week. She was then perfectly well for two weeks until pain and fever recurred on the trip to Australia. This attack lasted a week, and was followed by an apparent complete recovery. After two weeks, however, she had recurrence of the familiar prostration and fever and was under the care of Dr. Hooper, of Melbourne, for a time. A blood examination, made October 24, 1905, by Dr. F. W. Wilkinson, showed red corpuscles, 4,600,000; white corpuscles, 8000; hemoglobin, 90 per cent.; no malarial parasites; a distinct increase of large mononuclear leukocytes.

After this attack she felt well, and was able to travel considerably about Australia. From October to February there were three similar periods of pyrexia, but she was not confined to bed. In February there was a severe attack in Rotorua lasting a week, during which joint and muscle pains were intense. Since that time there has been absolutely no pain, and recurrent pyrexia has been the sole feature (see the accompanying chart).



The course of the disease to August 28, 1906, when the patient died, is given in the subjoined table.

No. of attack.	Pyrexia.	Apyretic interval. (Approximate.)
1	7 days	14 days
2	7 “	14 “
3	10 “	not known
4	7 “	“ “
5	7 “	“ “
6	7 “	“ “
7	7 “	10 days
8	7 “	10 “
9	3 “	14 “
10	7 “	14 “
11	22 “	14 “
12	22 “	9 “
13	75 “	

During the first attacks aching in the muscles and joints was intense, and suggested dengue. In the intervals health was perfect, and the patient travelled, ate well, and nothing suggested a return of the trouble until fever recurred. The temperature rose rapidly, but not abruptly, and fell by lysis; during the intervals, when measured, it was slightly subnormal. With the recurrence of temperature, there was increased constipation, some bloating, malaise, and, in the later periods, great prostration, occasional chills, and great discomfort from sweating. Under Dr. Humphris' observation, the pulse was of good quality, from 74 to 90; quinine did not affect the temperature, and made the nervous symptoms worse; pyramidon, in 5-grain doses, was found to give much relief, with a quick drop in temperature and profuse sweating. Until July there was no evidence of cachexia, and the absence of symptoms and signs was in striking contrast to the monotonous daily temperature.

When seen July 20, the patient had a peculiar earthy color, not unlike that seen in liver abscess. There was fever of a marked remittent, intermittent type. Shaking chills occurred every three or four days. At the height of temperature, there was great restlessness, sometimes mild delirium, but no pain. Pyramidon was occasionally required to control temperature; 5 grains would be quickly followed by a drop of 3° to 5° and a drenching sweat. Even without pyramidon, profuse sweating was frequent.

There were slight changes at both apices, but no evidence of an active process. The chief thing of interest in the thorax examination was the curved line of liver dulness, reaching to the fourth rib in the mammillary and anterior axillary lines, to the angle of the scapula and down to the eleventh spine at the vertebral column. Despite the enlargement of the liver upward, which showed beautifully on an *x*-ray plate, there was at no time any evidence of enlargement downward. The area of splenic dulness was frequently a little large, but the spleen was never palpated in almost daily examinations over a period of four weeks. The heart was markedly dilated, particularly the left ventricle, and there was a systolic murmur over the precordium, with greatest intensity over the base. The pulse was slow in comparison to the temperature, averaging 80 to 100.

The abdomen was considerably distended, not tender, and in the last days a small amount of free fluid could be demonstrated. Superficial glands could never be palpated, nor were there glands to be felt in the abdomen or by rectum. The follicles of the tonsils and base of the tongue were not enlarged—in fact the tongue suggested the smooth atrophy of Virchow, but was not positive. There was extreme myosis, which had been noted for some years. The pupillary outline was irregular, and reaction to light was practically absent. There were no fundus changes, and nothing else in the nervous system to suggest syphilis. Pelvic and rectal examination was negative. The urine at different times showed a trace of albumin, and a few hyalin casts. Diazo reaction was constantly present, sometimes faint, and again well marked. In the last days urobilin was present in considerable amount, with a trace of bile. The stools were microscopically negative, and contained no occult blood.

Cultures in bouillon, made on two occasions, with blood from puncture of a vein gave no growths. Serum reactions with cultures of typhoid and paratyphoid bacilli, and Micrococcus melitensis were negative in dilutions of 1 to 50 and 1 to 20. Of principal interest in the blood counts (see the accompanying chart) was the moderate secondary anemia, with marked leukopenia and relative increase in the large mononuclear forms. Some of these cells were distinctly larger than the normal type.

Date.	Reds.	Whites.	Percentage.				Hb.	Remarks.
			Poly-nuclear	Small mono-nuclear	Large mono-nuclear	Eosino-philes.		
October 24, 1905	4,600.000	8000	......	......	In-crease.	.....	90	No plasmodia. Made by Dr. John F. Wilkinson, Melbourne.
May 2, 1906	5,800,000	7200	78.0	21.0	1.0	......	...	No plasmodia. Made by Dr. Humphris, Honolulu, T. H.
May 21, 1906	5,600,000	5800	67.0	32.0	......	1.0		
July 21, 1906	4,416,000	3000	83.0	8.0	9.0	......	70	Temperature 102°. Slight variation in size of red cells.
July 24, 1906	......	5500	72.5	12.0	15.5	......	...	After chill. Temperature 104°.
July 28, 1906	4,032,000	2800	65.0	15.5	19.5	......	...	No plasmodia. Few degenerated reds.
August 4, 1906	3,100,000	5200	73.0	9.0	18.0	......	70	5 P.M., after chill. Temperature 103.6°.
August 11, 1906	3,272,000	2600	80.0	9.0	11.0	......	60	No nucleated reds.

With the peculiar recurrent pyrexia, the conditions considered in diagnosis were chiefly Malta fever, Hodgkin's disease or lymphosarcoma, syphilis of the liver, and gallstones in the common or hepatic ducts with catarrhal cholangitis. The aching of joints and muscles in the early periods, the undulant fever, the recent visit to many Mediterranean ports would suggest Malta fever, but the frequent repetition of attacks and the negative serum reaction decided against it. Serum reactions in this disease are not always unequivocal.[2]

Echinococcus of the liver was suggested by the shape of the right lobe, but the radiogram gave no characteristic picture, and the absence of eosinophilia and leukocytosis spoke against it.

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[2] An army pathologist of wide experience sent a man to the hospital with the diagnosis of Malta fever from the agglutination test; but the recurrent fever later cleared up on drainage of an unrecognized empyema. In another case with positive serum reaction, gonorrheal infection was shown to be the cause of temperature and of joint symptoms.

The absence of jaundice was much against a ball-valve calculus in the common duct. The occasional complaint of distress over the liver, with a feeling of distention in the abdomen, suggested the possibility of a calculus in the hepatic ducts, as in the cases of Frerich[3] and Schmitz.[4] The failure of a polynuclear leukocytosis just after a chill and rapid rise of temperature, was decidedly against an infective cholangitis. My experience has been that of Pick,[5] that in cases of infective cholangitis (of pyelitis and nearly all cases of malignant endocarditis as well), there is a decided polynuclear leukocytosis just after a paroxysm of chill and fever. There may have been a normal count, or a leukopenia, with normal ratio, during the febrile or afebrile period before the paroxysm, and the sudden change in the blood picture is most striking.

Despite the characteristic alternating pyrexia, the absence of palpable glands and spleen, and of enlarged mediastinal glands, was much against Hodgkin's disease or lymphosarcoma limited to the liver; although the cases of Naunyn[6] and Rolleston[7] had lately been brought to my mind in reviewing a case of intermittent hepatic fever.[8]

Naunyn mentions the case of a woman of forty with intermittent fever for some months and paroxysms of chill, fever, and sweating recurring about 4 P.M. daily. There was considerable liver enlargement and an irregular tumor of the left kidney. The patient demanded operation on account of the unsupportable chills and sweating. The left kidney was removed, and contained a sarcomatous mass the size of an apple. The liver appeared normal. The chills and fever ceased, and the patient improved until, after four weeks, the fever recurred. Death occurred from exhaustion, and at autopsy metastases were found in retroperitoneal and medistinal glands, and a large central mass in the liver.

The history of syphilis, the pupillary changes, the signs of some liver affection, demanded specific therapy. Mercury was given by inunctions and hypodermically, and iodide was administered up to 6 grams daily in combination with ascending doses of Fowler's solution. There was no improvement at the end of three weeks; the patient was weaker, slight icterus developed, and moderate ascites could be demonstrated. Operation was demandad by the patient for relief of the intolerable discomfort caused by the temperature and sweating, and an exploratory laparotomy was done by Dr. Beverly MacMonagle in the hope of finding an abscess, or, as in Israel's case, a gumma that could be reached surgically. On opening the abdomen a moderate amount of blood-stained fluid escaped. The liver edge was a little above the costal margin, was irregular, rounded, and the liver was studded with whitish, irregular nodules the size of a pea or a small marble. A piece of the liver was removed and sent to Dr. William Ophüls for examination. The wound was closed, and the patient sent to bed in fair condition, but death occurred next day from exhaustion.

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[3] Leberkrankheiten, Band ii, 495.

[4] Berl. klin. Woch., 1891, 915.

[5] Zur Diagnostik der Infekte der Gallenwege, Mittheil. aus den Grenz., d. Med. u. Chir., viii, 174; Ueber Intermittirendes Gallenfieber, Deut. Arch. f. klin. Med., lxix, 1.

[6] Cholelithiasis, S. 69.

[7] Diseases of the Liver, p. 388; Ibid. p. 152.

[8] Rolleston in writing of lymphosarcoma of the liver records that: "Exceptionally the organ is very considerably enlarged, and if the superficial lymphatic glands available for clinical examination are but little affected, the clinical aspect of the case, especially when there is a hectic temperature, may suggest hepatic abscess, as in a case under my care." Again, "Some years ago a man was under my care with a pedunculated mass in the left groin of many years' standing, which turned out to be lymphadenoma; he developed a hectic temperature and considerable enlargement of the liver. The condition was not unlike hepatic abscess, and the question of operation was raised during life."

The following is Dr. Ophül's report: "Sections show multiple tumor nodules, which, in the periphery, consist almost entirely of very large round or oval cells. Many of these cells show mitotic figures. The central parts of the nodules are made up of somewhat smaller cells, with some connective-tissue fibers between them. No caseation. In the periphery the tumor infiltrates the liver tissue following the course of the lymphatics along the capillaries. No definite alveolar arrangement can be made out in any place. The tumor has the appearance of a sarcoma."

At the autopsy the lungs were found emphysematous, with puckered scars and shrinking at both apices. The heart was flabby, much dilated, the endocardium normal, the ascending aorta dotted with atheromatous patches. Near the hilum of the right kidney, which was in normal position, was a large retroperitoneal gland the size of a golf ball. This was the only gland enlarged anywhere in the body, except a few pigmented glands the size of small beans, such as one usually sees about the roots of the lungs. The follicles of the gastro-intestinal tract were not enlarged. Chiefly remarkable was the enlargement of the dome of the liver, as had been determined clinically; the diaphragm was pushed up to the third rib. The failure of the liver to enlarge downward was explained by the puckering of the rounded edges by several deep scars and the presence of several firm adhesions to the anterior abdominal wall—plainly the result of an old luetic process. Great numbers of whitish nodules studded the right lobe of the liver; there were only five or six in the left lobe. The spleen was not enlarged, 12 by 7 cm.; on section there were three whitish, irregular nodules the size of lima beans similar to the growths in the liver. Microscopically the tumors of the lymph gland, liver, and spleen showed a similar structure to the nodule removed at operation. There was no caseation. Giant cells and eosinophiles were not present. No tubercle bacilli were found in the smears from the gland tumor. The growth would be best classed, therefore, as a lymphosarcoma.

Retrospectively, it would seem that the attacks of abdominal pain in 1904 marked the beginning of the disease, probably in the retroperitoneal gland, and that the fever, general pains, and indefinite symptoms in the hepatic region of the last year signalled the invasion of the liver. It is difficult to assign a definite role in etiology to syphilis, nor can the limitation of the malignant process to one lymph gland be in any way explained. Syphilis may perhaps have rendered the liver less resistant to invasion of the growth. Treatment would probably have availed little at any stage of the disease. Large doses of arsenic and specific treatment were given during the last month of life without effect. *X*-ray treatment and Coley's serum might have been tried in the apyretic intervals. *X*-ray treatment was out of the question while the patient was under my care, owing to the pyrexia and cachexia; it is, even in afebrile cases, at times distinctly detrimental.[9]

Recurrent fever in tuberculous lymphomas, in Hodgkin's disease, in lymphosarcoma or sarcoma of other varieties has been chronicled in numerous papers: Dr. Musser reviewed the literature, and added two personal cases in a communication to this Association in 1901. Graves mentioned occurrence of the peculiar temperature in some cases of lymphomas before Murchison's[10] paper in 1870. Gowers[11] introduced the name alternating pyrexia in his article on Hodgkin's disease in Reynold's *Practice*; Southey (1875) and Sainsbury were among the early English authors recording cases; Pel[12] in 1885, and Ebstein[13], in 1887, called general attention to the peculiar fever type, and Ebstein thought he was describing a new clinical entity, which he termed "das chronische Rückfallsfieber." It soon became recognized, however, that recurrent fever is a symptom and not a disease. The papers of Graves, Murchison, Southey, Gowers, Sainsbury, Pel, and Ebstein dealt with Hodgkin's disease. In Klein's[14] case, reported as cirrhosis of the liver and pseudoleukemia with recurrent fever, the history suggests strongly that fever was of the intermittent hepatic variety. Two of Kast's[15] cases would be classed clinically as Hodgkin's. In a boy of seven, with enlarged glands, spleen, and liver, there were nine periods of pyrexia, and toward the last a leukocytosis (the kind not described) of 100,000, and later, 270,000. Grawitz mentioned a case with enlarged glands becoming softer during the attacks of fever, negative blood cultures, and no bacteria in the glands at autopsy. Janz[16] extirpated the spleen in a case reported as pseudoleukemia; streptococci were found in many glands and tubercle bacilli in one supraclavicular gland. Fischer,[17] reporting 12 cases of Hodgkin's disease, in which cultures from the blood and glands were negative, mentioned one instance with periodic fever in which organisms were found in the blood during the pyrexia, but not in the intervals. Laache[18] saw Hodgkin's disease with alternating pyrexia, and noted decrease in size of the glands during the fever and increase in the apyretic intervals. He mentions that Fiedler reported recovery in a case of Hodgkin's with recurrent fever. The communications of Van der Scheer,[19] Barbrock,[20] Kissel,[21] Westphal, Verdelli,[22] Freudweiler,[23] likewise deal with Hodgkin's disease or "pseudoleukemia," as do recent artcles by Hirschfelder[24] and Clarke, Stoll,[25] Ruffin,[26] and Tschistowitsch.[27]

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[9] In an old man with generalized Hodgkin's disease, who had previously had periods of pyrexia, a few *x*-ray exposures were followed by remittent fever, which persisted till death some three weeks later. A man aged forty years had recurrence of lymphosarcoma in the spleen and liver six months after a primary tumor in the glands of the right axilla was removed. Four attempts were made at *x*-ray treatment. Each time, after three or four short exposures over the liver and spleen, there would be a chill followed by rapid rise of temperature to 103° or 104°, prostration, loss of appetite, and persistence of fever for four or five days.

[10] Trans. Path. Soc. Lond., 1870, xxi, 372.

[11] Reynolds' System of Medicine, 1879.

[12] Zur Symptomatologie der sogenannten Pseudoleukämie. Berl. klin. Woch., 1885, 3. Pseudoleukämie oder chronisches Rückfallsfieber. Berl. klin. Woch., 1887, 644.

[13] Das chronische Rückfallsfieber. Berl. klin. Woch., 1887, 565.

[14] Ein Fall von Pseudoleukämie nebst Lebercirrhose mit recurrierendem Fieberverlauf. Berl. klin. Woch., 1890, 712.

[15] Ueber Rückfallsfieber bei multipler Sarkombildung.

Shortly after Ebstein's paper it was shown that "chronisches Rückfallsfieber" might occur as a striking symptom of malignant disease. Renvers[28] reported lymphosarcoma of the retroperitoneal glands, liver, and spleen, marked clinically by recurrent pyrexia in seven periods of seven days each. In Voelcker's[29] case, with typical undulant fever, there was a sarcoma of the lumbar spine and retroperitoneal glands. Hanser[30] reported ten cycles of fever with an epigastric tumor regarded as sarcoma. In Hewelke's[31] case of retroperitoneal lymphosarcoma, staphylococci were found four times in blood cultures taken during both febrile and afebrile stages. Multiple myelomas were found as the cause of fever recurring at intervals of one to twelve days over a period of nine months in a case recorded by Hammer.[32] Subohn[33] reported a questionable instance of recurrent fever with lymphosarcoma of the thymus, and Witthauer[34] noted periodic fever through five months in a round-cell sarcoma of the posterior mediastinum. Kast, beside the 2 cases mentioned above, described recurrent fever in a sarcoma of the hip and retroperitoneal glands occurring in a boy of sixteen. The papers of Puritz,[35] Bulubasch,[36] Kissel, Glaeser,[37] Hohenemser, deal also with the association of alternating pyrexia and sarcoma. Hampeln,[38] Anker,[39] Kobler,[40] Kast and Freundweiler write of its occurrence with carcinoma.

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[16] Zur Operation der leukemischen Milz. Beitr. z. klin. Chir., 1899, xxiii, 287.

[17] Ueber malignes Lymphom. Arch. f. klin. Chir., 1897, lv, 467.

[18] Ebstein und Schwalbe's Handbuch, i, S. 844.

[19] Een geval van pseudoleukemie med gedeeltelyk recurrend Koorstype. Neederl. Weekblatt, 1899.

[20] Ueber Pseudoleukemie mit intercurriendem Fieberverlauf. Virchow und Hirsch's Jahresberichte, 1890, ii, 331.

[21] Maligne Neubildungen und chronisches Rückfallsfieber, Vratsch., 1895, No. 35; Drei Faelle von Hodgkinischer Krankheit bei Kindern Vratsch, 1895, 651.

[22] Archivo italiano di clinica medica, 1895.

[23] Statistische Untersuchungen über Fiebererscheinungen bei Carcinom innerer Organe. Deut. Arch. f. klin. Med., lxiv, 544.

[24] A Case of Hodgkin's Disease with Chronic Recurrent Fever. Occidental Med. Jour., 1904, xviii, 11.

[25] A Case of Hodgkin's Disease with Slight Enlargement of Peripheral Glands. Med. Record, 1905, p. 773.

[26] Hodgkin's Disease. A Study of a Case with Relapsing Fever. AMER. JOUR. MED. SCI., 1906, p. 587.

[27] Ueber Pseudoleukemie mit periodischen Fieber. Deut. med. Woch., 1907, 502.

[28] Ueber Lymphosarkomatose mit recurriendem Fieberverlauf. Deut. med. Woch., 1888, 753.

[29] Ueber Sarkom mit recurriendem Fieberverlauf. Berl. klin. Woch., 1889, 796.

[30] Ein Fall von chronischem Rückfallsfieber. Berl. klin. Woch., 1889, 692.

[31] Lymphomatosis retroperitonealis maligna cum febre recurrente Kronika lekarska, 1889, xii.

Delafield[41], and a little later Askanazy[42], first called general attention to the similarity of many cases of tuberculosis of the lymphatic system and Hodgkin's disease. Musser's[43] 2 cases of recurrent fever were tuberculous. In No. 8 of Sternberg's[44] series, this type of fever was noted, but the cases should be classed as Hodgkin's disease with terminal tuberculous infection. Schur[45] reported a combination of Hodgkin's disease and tuberculosis, with alternating pyrexia and a polynuclear leukocytosis of 30,000. Crowder described a case of generalized tuberculosis with irregular fever, a leukocytosis of 19,000, and the clinical picture of Hodgkin's disease; tubercle bacilli and staphylococci were found in the glands at autopsy. Strauss[46] saw a female patient with enlarged cervical glands and chronic recurrent fever. At one time there was such a marked lymphocytosis as to suggest lymphatic leukemia, but tuberculous glands were found at operation.

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[32] Primare sarkomatöse Ostitis mit chronischem Ruckfallsfieber. Virchow Arch., 1894, 137, 280.

[33] Jahrbuch der Hamburger Staatskrankenanstalten Leipzig, 1892, 65.

[34] Intermittirendes Fieber als Symptom eines Mediastinaltumors. Munch med. Woch., 1901, 224.

[35] Ueber Sarkom mit sogenanntem chronischem Ruckfallsfieber. Virch. Arch., Band 126, 1312.

[36] Sarkom der inneren Lymphdrusen mit febris recurrens. Medicinskoe obrosenie, 1894, Band 40-41, 601.

[37] Mit recurriendem Fieber verlaufende multiple Sarkomatose. Med. allg. Centralzeitung, 1897, 601.

[38] Zur Symptomatologie occulter visceraler Carcinome. Zeit. f. klin. Med., viii, 221; Ueber intermittirendes Fieber im Verlauf des Magencarcinoms. Zeit. f. klin. Med., Band xiv, 566.

[39] Ueber das Vorkommen intermittirendes Fieber bei chronischen Krankheiten. Inaug. Diss. Strassburg, 1890.

[40] Ueber typisches Fieber bei malignen Neubildungen des Unterleibs. Wien. klin. Woch., 1892, No. 23, 335; No. 24, 352.

[41] A Case of Acute and Fatal Tuberculosis of the Lymphatic Glands. Med. Record, 1887.

[42] Tuberculöse Lymphome unter dem Bilde febriler Pseudoleukemie verlaufend. Ziegler's Beit., 1888, iii, 413.

[43] Note on the Fever of Hodgkin's Disease. Amer. Med., 1902, iii, 13.

[44] Ueber eine eigenartige unter dem Bilde der Pseudoleukemie verlaufende Tuberculose des lymphatischen Apparats. Zeit. f. Heilkunde, xix, 21.

[45] Zur Symptomatologie der unter dem Bilde der Pseudoleukemie verlaufenden Lymphdrüsentuberkulose. Wien. klin. Woch., 1902.

[46] Sarcomatose und lymphatische Leukemie. Char. Annalen, xxiii, 343.

Although resting on different pathological bases, the clinical features of all these cases are strikingly similar. Febrile periods of from three to seven or ten days alternate with afebrile intervals of the same or slightly longer duration. As the disease progresses, the febrile periods usually lengthen, and continued fever of a remittent type may be present for weeks or months. The last attack in the case here reported was of seventy-six days' duration. The temperature usually rises and falls slowly, but there may be an abrupt rise with or without chill. Sweating is not uncommon. In the intervals between the first few febrile attacks the patients may feel and look perfectly well, but weakness, anemia, and cachexia gradually appear. Fever is frequently the fisrt symptom. The onset may be sudden, as in one of Pel's cases. Pain in the abdomen, as an early symptom, was noted by Ebstein, Hanser, and Voelckers. It occurred in three distinct attacks in the case here described. In Hammer's case of multiple myelomas, pain in the chest was the first symptom. A patient of Musser had severe pain in the right inguinal region with each pyrexial attack.

Pel, Ebstein, Bulubasch, Klein, and Tschistowitsch described yellowish discoloration of the skin with patchy, brownish pigmentation. Terminal icterus was noted by Pel, Ebstein, Musser, and Klein. Enlargment of the peripheral glands was absent in not a few cases (Pel, Ebstein, Renvers, Barbrock, Voelckers, Hanser, Tschistowitsch). The retroperitoneal and bronchial glands are more frequently involved. Absence of splenic tumor is not recorded in any case, except the one here noted. Spleen, liver, and palpable glands nearly always increase in size during the periods of fever, and frequently become tender. Laache noted a swelling of the glands in the afebrile periods and decrease of size with pyrexia.

The urine showed no characteristic changes. Albumin was infrequent, urobilin and bile pigment were occasionally noted. Diazo reaction was noted by Renvers, Stoll, and in my case. Examination of the blood helps in the diagnosis chiefly by ruling out other causes of fever. Secondary anemia becomes marked as the disease progresses. Stoll recorded a count of 1,700,000 red cells and 30 per cent. hemoglobin. Leukopenia is more common than leukocytosis. Polynuclear leukocytosis would speak more for tuberculous lymphomas than for Hodgkin's disease or lymphosarcoma. Strauss, however, noted a marked lymphocytosis in a tuberculous case, and a leukocytosis of 270,000 was recorded by Kast in a boy of seven. Relative increase of the large mononuclear cells has been described in several instances. It must be remembered, in differentiating from septic affections, that positive blood cultures have occasionally been reported. Positive or suggestive evidence of syphilis was found by Pel, Klein, Renvers, Voelckers, Tschistowitsch and myself. In 27 instances in which sex was recorded, there were 16 males and 11 females. Recorded ages varied from six to fifty-nine years. It is interesting to note that a number of patients had travelled extensively in the tropics. Recovery was noted in but 2 cases. Laache mentions a report by Fiedler and Tschistowitsch observed prompt cure under iodide after many other drugs had been tried for months. Death has occurred in all other cases, usually from exhaustion.

Recurrent attacks of fever may occur over long periods of time in many other conditions, and differential diagnosis is by no means always easy. Repeated relapses in typhoid fever will not often give difficulty; blood cultures will help in the cases in which Widal reaction is absent or long delayed. Claisse[47] has lately written on typhoid fever with numerous relapses. In my opinion, great hesitation should attend the diagnosis of sporadic cases of Malta fever. Piroplasmosis should be considered in all cases of continued fever from the tropics, and examinations made from splenic punctures if necessary.

Hampeln was the first strongly to emphasize the occurrence of fever in carcinoma. The papers of Kraussold and Max Freudweiler are more recent. Fever may be an early symptom; in a case of Hampeln there was fever for three months before a diagnosis was made. Continued fever is rare, and the remittent, intermittent, or hectic type the most common. Periodic attacks may be noted and ague-like paroxysms of chills, fever, and sweating. In 475 cases of carcinoma collected by Freudweiler temperature over 38° C. was present in 24.6 per cent. The carcinoma may cause fever even without ulceration: in 14 out of 64 cases reported by Freudweiler there was no ulceration.

Heubner[48] mentions fever recurring in eight periods during the fall and winter in a child of four. At first there were only general symptoms during the febrile attacks, but later pus appeared in the urine, and the diagnosis of pyelitis was assured. The frequency of pyelitis both in children and adults needs constant emphasis. It must be remembered also that renal calculi may rarely give rise to recurrent paroxysms of chills, fever, and sweating without any urinary signs; the attacks are similar to those of intermittent hepatic fever. "The attacks may occur, first, in tuberculous pyelitis; second, in calculous pyelitis; and, third, in rare instances of stone in the pelvis, without chronic suppurative pyelitis." (Osler.)[49]

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[47] Fievre typhoide prolongée avec rechutes multiples. Bull. et mém. Soc. méd. des Hôp. de Paris, 1906, xxiii, p. 571–574.

[48] Ueber langdauernde Fieberzustände unklaren Ursprungs. Deut. Arch. f. klin. Med., lxiv, 45.

[49] On Fever of Hepatic Origin, Particularly the Intermittent Pyrexia Associated with Gallstones. Johns Hopkins Hosp. Reports, vol. ii, p. 1.

Some years ago a woman aged forty-five years was under observation for recurrent fever of a years' duration. With periodic temperature, there would be occasional chills and

Romberg[50] mentions 2 cases with anemia, enlargement of the spleen and liver, and fever of months' duration—in 1 case of continued type; in the other with apyrexial intervals of weeks' duration. A mitral murmur was interpreted as of functional origin, and both cases went to autopsy with a diagnosis of Hodgkin's disease. The postmortem revealed malignant endocarditis. Fever and progressive weakness and anemia may be the only symptoms of infective endocarditis. There may be apyretic intervals of days and weeks at a time. The interpretation of heart murmurs in cases with high temperature and anemia is, as Bloch[51] has pointed out, often peculiarly difficult. Infective endocarditis grafted upon an old valvular lesion may run an extremely chronic course, over a year (Dreschfeld), thirteen months (Osler), and fifteen months (Romberg). Cases under my observartion with autopsy verification have lasted six, fifteen, and eighteen months. With absence of signs of heart involvement, diagnosis of the condition may be difficult or impossible. Cases of this kind have recently been reported by Cole[52] and Bramwell.[53] In chronic cases, leukocytosis may not occur; repeated counts in a case lasting fifteen months showed a leukopenia of 3000 to 5000, with a relative lymphocytosis. Ewing[54] holds that "leukocytosis is present probably in all cases, but is often intermittent, and single observations may fail to discover it." There is frequently a decided increase in the polynuclear count after a chill, and abrupt rise of temperature. Blood cultures are of great help in diagnosis.

Frequent mention has been made of intermittent hepatic fever. Pain in the region of the liver and jaundice deepening after paroxysms of chill and fever usually make diagnosis reasonably certain. In a case seen some years ago there was no jaundice until eight months after fever was noted. The increase of leukocytes, after chill and fever, as described by Pick has been twice observed. Pain and jaundice may be absent, and the temperature curve the only characteristic feature. Frerichs has described long-continued intermittent fever with stone in the hepatic duct, and Schmitz reported 2 cases of recurrent attacks of intermittent

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sweats, and complaint of dull aching in the right hypochondrium and lumbar regions. Leukocytes were normal in apyretic intervals, but after a chill and abrupt rise of temperature, there was several times noted a polynuclear leukocytosis of 28,000 to 36,000. There was a history of hematuria some years before, but the urine never contained albumin, blood or more than a few leukocytes. Operation revealed a large stone in the pelvis of the right kidney with a catarrhal pyelitis, no complete blocking of the ureter and no pyelonephritis. The fever with intermittent pyonephrosis (especially in tabetics) is, in my experience, frequently misinterpreted.

[50] Krankheiten der Kreislaufsorgane. Ebstein und Schwalbe, Handbuch der Praktischen Medizin, i, 685.

[51] Ueber Blutkrankheiten unter dem Bilde der akuten Endocarditis. Deut. med. Woch., 1903, 512.

[52] Johns Hopkins Hosp. Bull., xiii, p. 252.

[53] Clin. Studies, Edin., 1905–1906, n.s. iv, p. 193–207.

[54] Clinical Pathology of the Blood, p. 360.

pyrexia without pain or jaundice, in which cessation of fever followed passage of gallstones.

Many interesting papers of late have dealt with fever in visceral syphilis. Janeway[55] presented a communication on "Fever in Tertiary Syphilis" to this society in 1898. Riedel[56] cites remarkable cases in his article "Ueber die fieberhaft veraufende Lues der Gallenblase, Gallengänge, sowie der Leber," and quotes "den alten Satz, dass man bei unklaren fieberhaften Krankheiten immer zuerst an Tuberculose, so dann an Lues, endlich an Aktinomykose denken soll." F. Kemperer[57] reports several cases of syphilis of the liver with fever; in 1 case there was alternation of pyrexia with afebrile intervals over a period of a year. Recent literature on the subject is given in the articles of Klemperer and Raubitschek.[58]

In the past year Gilman Thompson called attention to the similarity between some cases of exophthalmic goitre and infective endocarditis. Two years ago an army captain was under my observation for some months. The chief complaint was of weakness, irritability, with occasional attacks of diarrhœa, chills, fever, and sweating. There was cachexia, a mitral systolic murmur, a pulse of 120 to 140, and irregular temperature recurring at intervals of days or weeks. The case was regarded as one of malignant endocarditis, until tremor, the general nervous picture, and Graefe and Stellweg signs decided for Parry's disease, even with absence of exophthalmos and goitre. A goitre developed later, and an operation by Dr. Halsted led to great improvement.

It is not uncommon to see children or young adults with long-continued or, more rarely, recurrent pyrexia, dependent upon tuberculosis of mediastinal, mesenteric, or retroperitoneal glands. A boy of eight was seen a year ago who had had moderate fever almost daily for six months. There was a history of pertussis one year before and examination showed enlarged bronchial glands. A young woman, seen four years since, had, as chief complaint, pain in the lumbar spine and in both loins. There was recurrent pyrexia of moderate degree over a period of eight months without satisfactory explanation. A radiogram showed the spine normal, but gave the shadows of several calcified retroperitoneal glands.

In a remarkable case, seen three years ago, a young woman of tuberculous family, had had recurrent pyrexia for over twelve years. Fever recurred two or three times in one year, and again not for two or three years. There were no symptoms during the attacks beyond prostration and temperature of 103°

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[55] Fever in Tertiary Syphilis. Trans. Assoc. Amer. Phys., 1898.

[56] Ueber die fieberhaft verlaufende Lues der Gallenblasse, Gallengänge, sowie der Leber. Mittheil. aus den Grenz., xi, 1.

[57] Ueber Fieber bei Syphilis der Leber. Deut. Zeit. f. k. Med., lv, 176.

[58] Zur Kenntnis der fieberhaften tertiär-syphilitischen Organ-Erkrankungen. Centralbl. f. d. Grenz., ix, p. 641.

to 105° for two or three weeks. During several attacks the cervical glands had enlarged and, when personally seen, there was evidence of bronchial gland enlargement. The liver and spleen were not palpable and the blood count was normal. Death occurred two years later from puerperal infection, but, unfortunately, no autopsy was obtained.

As seen above, alternating pyrexia may be a symptom of many very different condititons. Regularly repeated attacks over long periods of time are most suggestive of tuberculous lymphomas, Hodgkin's disease, lymphosarcoma or other varieties of sarcoma. In any recurrent pyrexia, the possibility of disease of deep glands should be remembered. Rarely, as in the case recorded by Rolleston, or in the one here described, the liver may be, clinically, the sole organ affected.

TRUE INTESTINAL DYSPEPSIA.[1]

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By the expression "intestinal dyspepsia" we understand conditions in which the digestive function of the intestinal tract is disturbed. The idea of testing the functions of the intestines in a manner similar to those of the stomach prevailed among clinicians for a long while, but its execution was attended with great difficulties. To Adolf Schmidt[2] belongs the credit of having elaborated a method to that effect. This well-known clinician introduced the test diet, known under his name, for the purpose of examining the feces. By means of this method, Schmidt and Strasburger[3] were enabled to discover a group of cases of intestinal dyspepsia, in which the starch digestion is particularly disturbed. They designated it fermentative dyspepsia.

Schmidt[4] has recently described the occurrence of functional achylia of the pancreas; by this he understands cases in which there is a malassimilation of the food, and a temporary lack of nuclear digestion—assigned by him as characteristic of the pancreatic secretion.

In a former paper[5] I have described similar cases, in which all nutritive substances were poorly digested. Yet I hardly believe it advisable to speak of achylia of the pancreas, because by achylia we understand a complete absence of secretion. The conditions in the bowel are very complicated, depend upon many factors, and it is very difficult to judge which organ is at fault. However interesting the nucleus test may be, I do not think that it is of very great value in the recognition of disease of the pancreas, and I fully agree with Steele[6] when he says: "My clinical experience with the nucleus test does not entirely support Schmidt's claim for the method." I have only recently observed a case of tumor of the liver, with chronic jaundice, in which the nuclei at first were unchanged, but later—shortly before death—disappeared entirely. On the other hand, I have observed a case of achylia gastrica and grave anemia in which the pancreas was not involved, and in which the nuclear digestion was almost entirely absent. Besides, in all the cases of non-disappearance of the nuclei, it is possible that the pancreatic secretion is present, but its action inhibited by the presence of some other noxious substance (HCl, organic acids, etc.). It is, therefore, better to classify these cases under the heading of intestinal dyspepsia.

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[1] Read at a meeting of the American Gastro-enterological Association, at Atlantic City, N. J., June 3, 1907.

[2] The Test Diet in Intestinal Diseases. Translated by Charles D. Aaron, Philadelphia, 1906.

[3] Deut. Arch. f. klin. Med., 1901, lxix, 570.

[4] Functionelle Pankreasachylie, Deut. Arch. f. klin. Med., 1906, lxxxvii, 456.

[5] Further Remarks on the Digestive Bead Test for Ascertaining the Functions of the Digestive Apparatus. Jour. Amer. Med. Assoc., February 2, 1907.

In the application of Schmidt's test-diet there are, however, some difficulties. It is often necessary to wait two to three days for the stool from the test-diet, and frequently the results are not easy to interpret. For these reasons Schmidt's method has not been adopted in general practice.

The bead test is easy to apply, and its results are sharply defined. Through a large number of examinations, which will soon be published, I have become convinced that the digestive functions of the bowel may be well judged by means of this test. There is, therefore, no reason why we should not utilize the test in the study of diseases of the bowel.

It is advisable to distinguish two large groups of intestinal disturbances: (1) Digestive disturbances which concern all three classes of food substances (albuminates, carbohydrates, fat): "dyspepsia intestinalis universalis." (2) Digestive disturbances affecting only certain classes of foodstuffs: "dyspepsia intestinalis partialis." This second group may be subdivided according to the varieties of foods, the digestion of which is disturbed: (*a*) Disturbed digestion of proteids; (*b*) carbohydrates; (*c*) fats; and (*d*) two classes of foods, namely, proteids and carbohydrates, carbohydrates and fats, and proteids and fat. These functional disturbances of the intestinal digestion may exist without appreciable anatomical change of the digestive apparatus, or they may be associated with lesions of the intestine. Occasionally subjective disturbances are found in the intestinal tract, in which neither anatomical nor functional deviations from the normal occur. These may be considered as true nervous intestinal dyspepsia.

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[6] The Nuclear Test in Pancreatic Disease. Univ. Penna. Med. Bull., November, 1906.

In using the bead test, we must not imagine, if we find a substance in the beads undigested, that no digestion whatsoever of such substance may occur in the bowel. That would be wrong, for the same foodstuffs in liquid form may still be well digested. We can only conclude from this test that there is a considerable diminution of the digestive power for these substances.

I should now like to describe a few cases of functional intestinal dyspepsia which belong to both groups. The first two cases belong to group one (dyspepsia intestinalis universalis):

Case I.—*Achylia Gastrica and Dyspepsia Intestinalis Universalis.* Israel R., aged sixty-two years, has been suffering for several years from a loss of appetite and digestive disturbances. Lately his trouble has increased and he complains of pain shortly after meals. His appetite has entirely disappeared. Patient sleeps poorly and complains a great deal of obstinate constipation. He has lost somewhat in weight.

Examination of the chest reveals normal conditions; in the abdomen, also, nothing abnormal is found. Examination of the stomach contents one hour after a test breakfast shows absence of HCl and rennet and a total acidity of four; very little fluid, and particles of roll scarcely changed. We have to deal here with a marked case of achylia gastrica.

The bead test[7] was given, with the following result: 4 beads are found after twenty-four hours; 1 after forty-eight hours; meat, fat, fish-bone, thymus, potato present; catgut was not found; nuclei in thymus have disappeared. Two weeks later the bead test was repeated and showed the presence of meat, potato, fat, thymus, and fish-bone.

The patient was put on a diet of milk, koumiss, raw eggs, soups, and strained gruels, and pancreon was given tentatively. He felt somewhat better, did not lose in weight, but his troubles did not cease. One and one-half years later he was still in the same condition: poor appetite, disagreeable sensations and constipation; however, he has not lost weight since January, 1906.

Case II.—*Achylia Gastrica and Dyspepsia Intestinalis Universalis.* October, 1906. E. S. N., aged fifty-two years, complained for two years of digestive disturbances accompanied by severe attacks of diarrhœa and colicky pains. His appetite is moderate. For the last six months he has had constant diarrhœa and has lost about twenty pounds. A sojourn in a German watering-place has not given him any relief. Thoracic organs are normal. In the abdomen atony of the stomach is found. One hour after Ewald's test breakfast, a state of complete achylia is found. The bead test shows after twenty-one hours: thymus, meat, fish-bone, catgut, and half of the potato present. Nuclei and fat have disappeared.

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[7] Archiv f. Verdauungskrankheiten, Heft i, table i, case 17, xiii

The patient was placed upon a diet of milk, koumiss, eggs, and fine gruels, and was given codein with tannigen. He gained slowly in weight and the diarrhœa was checked. In seven weeks he had gained eight pounds. Another examination of the stomach contents, as well as a bead test, was made. The stomach showed complete achylia, and the in bead test thymus, meat, catgut, and potato were found—everything else having disappeared.

The patient is still in about the same condition. He has not lost during treatment, and is still suffering more or less from diarrhœa.

CASE III.—*Dyspepsia Intestinalis Partialis.* Mrs. S., about thirty-two years old, rather corpulent, and with the appearance of good health, has been complaining for about a year. She has a disagreeable taste in her mouth. There is a feeling of pressure and fulness in the entire abdomen. She frequently belches and passes wind. She is always constipated, and must take strong cathartics. Even then the stool is insufficient and of a very offensive odor. The appetite is poor; she suffers from vertigo and tires very quickly. She does not sleep well. Recently she has been losing in weight.

Physical examination reveals nothing abnormal. Examination of the stomach contents is refused. The bead test shows after forty-six hours: thymus, meat, and fish-bone are present; potato, catgut, and nuclei have disappeared; fat bead was lost. Microscopically the stool shows a great number of large muscle fibers and smells very badly.

CASE IV.—*Dyspepsia Intestinalis Partialis.* Mr. M. complains of a feeling of fulness in the stomach and abdomen, much belching, great weakness, and persistent constipation. Examination shows systolic murmur at the apex beat; stomach extends a hand's width below the navel; right kidney is movable in the third degree. The stomach contents, removed one hour after breakfast, show the presence of free HCl, and a total acidity of 80. The diagnosis, therefore, is mitral insufficiency, enteroptosis, and hyperchlorhydria.

Patient was treated according to the usual methods. His general health improved, he gained in weight, but the constipation as well as the disagreeable sensations in the stomach and abdomen remained. The bead test was applied. The bead string passed after twenty-eight hours and showed the presence of thymus, fat, and half of the potato. Catgut, meat, fish-bone, as well as thymus nuclei, had disappeared.

The patient received takadiastase 0.3, three times daily. He at once improved. The sensations in the abdomen disappeared and the stool became nearly normal by the use of mild laxatives. The improvement has been permanent, and patient gained considerably in weight and strength.

CASE V.—*Hyperchlorhydria and Chronic Enteritis.* May 1, 1907. Oscar G., aged thirty-eight years, merchant. Previous history negative. Six years ago he complained of gastralgia, at first over the appendix. Since then he localizes the pains somewhat higher (in the region of the gall-bladder). He took calomel and became worse, and at the same time the pains became more intense. These pains attack him now and again. He cannot breathe deeply when he has them. Between the attacks the patient feels a certain amount of discomfort in his abdomen, about two hours after meals. The bead test shows: beads regained after twenty-four hours—fat, half of the thymus and of the potato present. Nuclei and everything else have disappeared.

Case VI.—May 9, 1907. Mrs. W., aged thirty years, was always well until seven years ago when she was married. She has had two children, and was perfectly well when she was pregnant. Seven years ago she began to have trouble with her digestion. She belches considerably and has a great deal of wind, and her bowels are constipated; they may be normal for awhile, then they become constipated and directly after again loose. Appetite is poor.

Examination shows that the chest organs are normal; the stomach reaches one finger's width below the navel; the right kidney is slightly movable. One hour after test breakfast HCl=O, total acidity=20. The bead test shows: string regained after twenty hours—meat, potato, thymus (nuclei disappeared), are present; everything else has disappeared.

Case VII.—R. T. W., aged thirty-eight years, has been suffering for three years from severe attacks of pain in the upper abdominal region with subsequent vomiting. Between attacks the patient feels fairly well, but is very much constipated.

Examination revealed pronounced atony and ptosis of the stomach; otherwise nothing abnormal.

During an attack of vomiting free HCl and bile were demonstrated in the vomited matter. Ischochymia was not found. A week after the attack, when patient was feeling well, the bead test was used with the following result: after twenty-six hours—fat and potato are present; everything else had disappeared.

Epicrisis. Both cases in group I shows that complete intestinal dyspepsia may exist for a long time without necessarily endangering the life of the patient. In both patients achylia gastrica was present. I do not, however, doubt that occasionally, even in the presence of gastric juice, a complete intestinal diyspepsia may be found. Case II to VII of group II are good examples of various special disturbances of the intestinal functions. Disturbances of carbohydrate digestion seem to be most frequent, and those of meat digestion the rarest. Future clinical observation of numerous cases will determine the subjective signs of these various intestinal dyspepsias, so that from them alone the diagnosis may be conjectured.

Therapeutically we can gather a few valuable points as a result of the testing of the intestinal functions. For instance, in complete intestinal dyspepsia we must put the patient on a liquid or semiliquid diet and administer pancreon and similar medicaments. In partial intestinal dyspepsia we can limit the amount of that variety of food for which the particular disturbance exists. Takadiastase, in doses of 5 grains three times daily, has been a valuable help in cases of amylaceous intestinal dyspepsia, as it seems to have removed the subjective symptoms in several cases of this kind. Pancreon in doses of 5 grains in disturbances of proteid and fat digestion seems to work well, although the results here are not so marked as in the former group.

PAROXYSMAL TACHYCARDIA.

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The following study, based on 9 cases, is an attempt to show that in paroxysmal tachycardia we have to deal, not with a single pathological entity, but with different pathogenic forms.

Paroxysmal tachycardia is characterized by periods of extremely rapid pulse rate, of sudden onset and abrupt termination. It has been extensively studied by A. Hoffmann,[1] who showed that the increased heart rhythm is either double or four times the normal. Mackenzie[2] then showed that in some cases tricuspid insufficiency could be demonstrated during the tachycardia, while the heart action during the interval occurred in the normal auricular rhythm. Recently Schlesinger[3] has directed attention to the relation of paroxysmal tachycardia to pathological changes of the central nervous system.

The attacks are characterized by the sudden onset of tachycardia, its equally sudden termination, the absence, in the majority of cases, of pronounced subjective symptoms, except at the beginning and the end of the paroxysm. In 2 cases the attacks began with a feeling of chest-constriction analogous to the onset of a mild angina; in one other, choking and air-hunger were complained of; in the remaining cases distressing palpitation was the only sensation noted. Similar sensory disturbances, even more disturbing, marked the end of the tachycardia. The duration of the attacks was variable; the rapid pulse rate lasted in 1 case but ten to fifteen minutes at a time, while the longest attack lasted for five days.

From a study of the cases I am led to divide them into four etiological groups: (1) Cases of paroxysmal tachycardia occurring in patients with previously damaged heart; (2) cases of paroxysmal

[1] Ztschr. f. klin. Med., vol. 53.

[2] Study of the Pulse.

[3] Sammlung klin. Vorträge, 143.

tachycardia in patients affected by dysthyreosis; (3) cases of paroxysmal tachycardia in patients exhibiting central nervous lesions; and (4) cases of apparently functional character, so-called idiopathic group.

The following cases belong to the first group:

Case I.—M. W., a female, aged thirty-four years, consulted me on account of dyspnœa on exertion, faintness, and vertigo. She had suffered from a mild degree of hemopilia, and presented changes in the heart and circulatory system. The pulse was regular, 80 per minute; the vessels normal; no arteriosclerotic changes; blood pressure above 150 mm. The apex beat was in the sixth interspace, 1 cm. outside the mammillary line, heaving in character. The heart dulness was increased, extending 2 cm. to the right of the sternal border. The heart sounds, excepting the second aortic, which was booming in character and strongly accentuated, were normal. The urine, on very frequent examinations, contained no albumin or casts. The backgrounds of the eyes showed no changes. The heart changes were considered secondary to the high pressure. While under treatment she developed a febrile attack, which was followed by a series of paroxysmal attacks, the pulse rising from the normal 80 to 100, to 150 to 200 beats to the minute. The attacks lasted from ten to fifteen minutes, and recurred repeatedly in twenty-four hours. This series of attacks lasted for six weeks, and finally subsided under roborating treatment and small doses of digitalis continued during the entire illness. The paroxysmal attacks subsided entirely for more than a year, but returned with a new febrile attack.

We had to deal in this case with the occurrence of paroxysmal tachycardia in a heart weakened by the strain of a high arterial pressure. The strain of the infectious process on the heart led to the attacks of paroxysmal quickening of the heart action.

Case II.—C. M., aged fifty years, alcoholic, drinks beer to excess, formerly heavy smoker, present weight 230 pounds. His present sickness began about twelve years ago, when he suddenly dropped to the sidewalk on account of dizziness and violent throbbing in his chest. He was several months in a hospital where he had several attacks every day, lasting from fifteen minutes to six hours, during which the pulse frequently varied between 150 and 200 beats to the minute. Further series of attacks were brought on by drinking or over-exertion, and usually repeated themselves every second or third year. The present attack was brought on by the strain which the man was subjected to after the earthquake. The patient is very fat; slight exertion causes considerable dyspnœa. The pulse is regular and of low tension; the arteries are slightly thickened, but not calcified. The heart dulness extends 2 cm. to the right of the sternal border. The heart sounds are feeble and distant, the first is much shortened, and resembles the second. The second pulmonic sound is slightly accentuated; the second aortic normal. During the paroxysm the heart is not enlarged; the sounds have assumed the embryonic rhythm; over the tricuspid area a soft systolic murmur is perceived and transmitted along the liver border. The veins of the neck, which before the attack showed a presystolic pulse, became very distended and pulsated violently, synchronously with the radial pulse, and were, therefore, of the ventricular type.

Another case of the first group was repeatedly seen in consultation with Dr. Gray.

Case III.—Mrs. A., aged, forty-five years. Has been suffering from attacks of frequent heart action, which were first noticed with an attack of cholecystitis; the pulse frequency during the paroxysm attained 200 to the minute, with typical onset and termination. The first attacks were mild and did not interfere with her household duties. In the last two years, however, the attacks have become so frequent that even the slightest exertion causes paroxysms of dyspnœa and tachycardia, so that she has been unable to leave her sick room. At the time I saw the patient she was wasted and anemic. A few days previously a relapse of her cholecystitis had occurred. Her pulse was fairly regular, varying between 80 and 90. The heart extended 3 cm. to the right of the sternal border. A systolic murmur could be heard over the apex and was transmitted to the axilla; a second systolic murmur was heard over the tricuspid area and was propagated along the liver border. The veins of the neck showed a systolic pulse; the liver was distinctly pulsating. While we were examining the patient an attack started, the pulse rate changing suddenly to 140 to 154 to the minute. The pulse during the attack was irregular, seeming to consist of a series of extra systoles.

The interesting facts in this group are that the heart lesion alone does not cause the symptom-complex, which originates only when a new weakening influence interferes with the action of the heart. As long as the heart has not sufficiently recovered the slightest provocation will lead to a paroxysm. The attacks, therefore, will occur in series. With the recovery of the heart muscle the tendency to attacks diminishes and the completely compensated heart does not present any attacks.

In the second group the paroxysmal attacks seem to be due to dysthyreosis. A number of observations have been published in which paroxysmal attacks complicated an exophthalmic goitre (Hoesslin,[4] Warden,[5] and P. K. Brown[6]). I have observed the occurrence of paroxysmal tachycardia in a case of dysthyreosis, and in a case of Dercum's disease (adiposis dolorosa) reported by Dr. J. B. Frankenheimer. The patient seemed to be absolutely unaware of the rapid heart action. Whether this was due to her impaired mental faculites I could not decide.

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[4] Morbus Basedowii.

[5] Jour. Amer. Med. Assoc., 1906, p. iii.

[6] California State Med. Jour., 1906.

Cases falling in the third group have received particular attention during the last few years. Numerous publications have demonstrated the frequent occurrence of tachycardia in pathological changes of the central nervous system. Nothnagel[7] first called attention to the occurrence of paroxysmal attacks with epileptic seizures. Numerous analogous cases have since been recorded. Schlesinger has observed epilepsy in 25 per cent. of his cases of paroxysmal tachycardia. He, however, draws attention to the fact that all these cases belonged to the symptomatic group of epilepsy caused by anatomical lesions.

The occurrence of paroxysmal tachycardia has been described after injuries to the head; other instances occurred with tabes; a typical case, combined with multiple sclerosis, has been described by Müller,[8] at the postmortem of which an extensive involvement of the vagus nuclei was found. A lesion of the medulla oblongata caused by a luetic endarteritis of the basilaris was found to be present in a case of Reinhold.[9] In some cases, tumor of the brain constituted the lesion (Pitres).[10] Tumor of the brain involving the frontal and parietal lobes around Roland's fissure, and causing paroxysmal tachycardia, was described by Reinhold. A case belonging to this group is the following:

Case IV.—A. B., aged sixty-six years, formerly alcoholic. He entered the hospital on account of emphysema and œdema of the legs. The patient showed marked arteriosclerosis in all the palpable arteries. The heart and aorta were slightly enlarged. Over the apex there was a mitral systolic murmur which disappeared with the improvement of the condition. The lungs were emphysematous, with many rales. In the hands and arms a very marked tremor was present, consisting of movements of flexion and extension of the hands and the fingers, occurring at the rate of 7 to 8 to the second, present most of the time, even in absolute rest. If he got excited, or tried to execute a difficult movement, the tremor became much more pronounced and resembled the tremor of multiple sclerosis. No ataxia; no Romberg sign. No paralysis or paresis of any muscle, but the muscles all over the body were hypertonic, most marked in the right leg. No nystagmus. Face mask-like. Speech monotonous, but otherwise undisturbed. Babinsky of right foot and an increased knee-jerk pointed to a lesion of the pyramidal tract; the abdominal reflex was absent on the right side, very feeble on the left. No disturbance of the bladder or rectum.

We have to deal, in this case, with an extensive arteriosclerosis combined with lesions of the nervous system. These point to a non-systemic disturbance of the cord and brain. In many respects it resembles an atypical case of multiple sclerosis. The advanced age, however, and the existence of a marked arteriosclerosis point to the arteries as the cause of the nervous lesion (Oppenheim, Naunyn, Nonne, Luce, Demande). The attacks of paroxysmal tachycardia are probably due to a disturbance of the medulla oblongata, caused by the narrowing of the basilar artery, as in the case described by Reinhold.

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[7] Ueber parox. Tachycardie, Wien. med. Blätter, 1887.

[8] Multiple Sclerose, Jena, 1904.

[9] Ztschr. f. klin. Med., vol. lix.

[10] Arch. clin. de Bordeaux, 1894.

Some interesting cases in which the paroxysmal tachycardia was caused by a lesion of the peripheral nervous system have been described by Schlesinger and Pal.[1] In Pal's case, several branches of the sympathetic nerve were involved in an adhesion going from the apex to the right innominate vein; whether this lesion, the influence of which would consist in an excitation of the accelerans, accounts for the symptom complex, seems to me to be very questionable, as the anatomical position of the accelerans in man has so far not been determined.

In Schlesinger's case the most important lesion discovered at autopsy consisted of an inflammation of the right vagus. During life the paroxysmal attack could be very easily stopped by compression of the right vagus, while compression of the left vagus seemed to be without any effect on the attack. In connection with the right-sided neuritis of the vagus, this observation proves the intimate relation of this lesion with the symptom complex. The pathogenesis of the attacks, however, is not made any clearer by this finding. In other cases in which a compression of the vagus was found a permanent tachycardia resulted (cases of Riegel, Ortner, Stix, Bois, and Sommer). It still remains to be explained why in some cases this lesion leads to a permanent tachycardia, in other cases to the paroxysmal form.

To the fourth group of apparently functional character belongs the following observation:

CASE V.—J., aged sixty-eight years. Complains of attacks which, according to his conception, consist of a weakening of the heart action in so far as the heart has to contract twice to keep up the circulation. This conclusion he draws from the fact that his pulse rate doubles during the attacks. These began about twenty years ago, when the patient, who is a sea captain, got over-tired by a series of sleepless nights. The paroxysms usually begin with a feeling of constriction around the chest and choking; at the same time the pulse rate doubles; the attack lasts for a few minutes, and stops as abruptly as it started, while the pulse becomes slightly irregular for a few minutes. Deep breathing or pressure against the left side of the chest stops the attack. Sometimes he is free for several months, then again the attacks will come on several times

[1] Centralbl. f. innere Medizin, 1903.

a day. The examination shows that the heart is not enlarged, the blood pressure not increased; the kidneys work normally. The arteriosclerosis found is too slight to explain the tachycardia.

It seems that conditions in this disease are very similar to those seen in epilepsy, with which the paroxysmal tachycardia has numerous analogies, so many in fact that it has been considered as an equivalent. As in that disease, there are cases to be explained by an anatomical change of the nervous system, while in others the lesion is purely functional.

The mechanism of the attack was studied by tracings taken during normal heart action and during the paroxysms. This study leads to the conclusion that we have to distinguish two forms of tachycardia: (1) Forms in which the auricular wave is missing, the jugular pulse being of the ventricular form; and (2) forms in which an auricular wave appears in the tracing obtained during the tachycardiac rhythm. In these cases the ventricular systole follows an auricular contraction.

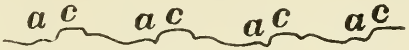

FIG. 1.—Normal venous pulse, showing auricular wave *a*, and transmitted carotid wave *c*.

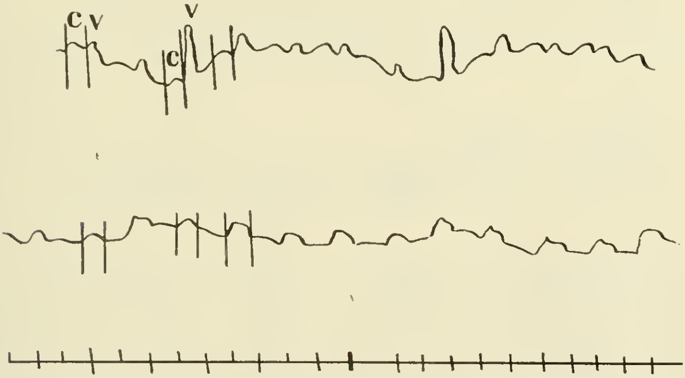

FIG. 2.—The upper tracing was obtained from the jugular vein, the lower tracing from the carotid artery; the time-markings correspond to one-fifth of a second. The tracing shows that the venous pulse commences synchronously with the carotid pulsation; that, therefore, during the attack the ventricular contraction is not preceded by an auricular contraction.

Fig. 1, a tracing taken during normal heart action, shows the usual form of a feeble auricular venous pulse, the wave *a* indicating the auricular contraction, *c* the transmitted carotid pulsation; the wave *v* is not pronounced in this tracing. The heart action is regular, 72 to the minute. During the attack the character of the pulse changes entirely (Fig. 2). The feeble pulse has become very marked and has changed to the ventricular form. The simultaneously registered carotid pulse shows that no contraction occurs before the beginning of the ventricular systole indicated by the upstroke in the arterial tracing. The venous pulse begins only with the ventricular systole and consists of the usual two waves of the normal ventricular venous pulse. With the cessation of the attack the ventricular venous pulse is again replaced by an auricular, while the veins collapse.

The explanation of this change is found in irregularities which occurred occasionally during the normal heart rhythm (Figs. 3 and 4). These are clearly due to the occurrence of extra systoles. The absence of the auricular wave in the extra systoles of Fig. 3 shows clearly that no auricular beat occurs before the extra systolic pulse. Therefore, the extra systole originates below the auricle either from the ventricle or the atrioventricular bundle. Fig. 4 demonstrates that during the extra systole the venous pulse shows exactly the same character as during the paroxysmal attack, and has changed to the ventricular type. The change of the venous pulse from the auricular to the ventricular form is, therefore, best explained if we assume that the paroxysmal attack is due to a series of extra systoles originating below the auricle. This view is corroborated by the comparison of the pulse rates. Fig. 5 shows the two different rates; the slow pulse rate was obtained by stopping an attack by deep inspiration. During the attack two systoles last 0.75 seconds, giving a pulse rate of 160. During normal heart action, taking immediately after the tachycardiac rhythm had stopped, a systole lasted 0.82 seconds, giving a pulse rate of 73 to the minute. We see that no relation exists between the two rhythms. The regularity of the rhythm during the attack is no objection to the extra systolic character of the attack, as the regular and rhythmical occurrence of

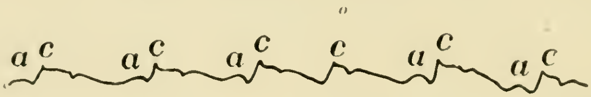

Fig. 3.—Venous pulse. At the point marked o, an extra systole occurs, in which the auricular contraction is missing.

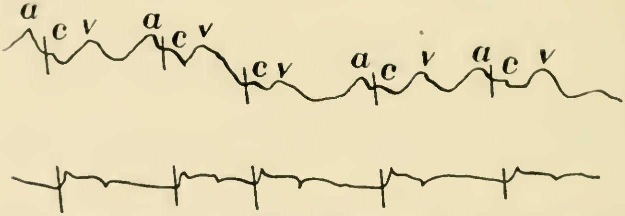

Fig. 4.—The upper tracing shows the venous pulse, the lower tracing, the carotid pulse registered simultaneously. At the point marked o an extra systole occurs without auricular contraction.

extra systoles has already been described by Pan[12] and Gebhardt.[13] The origin of the extra systole is indicated by the intermission following the extra contraction. Ventricular extra systoles may occur in two ways: either the pulse following the extra systole lasts until the next normal beat occurs, the time of the preceding systole plus the extra systole being equal to the time of two systoles; or the extra systole in a slow rhythm may occur without any disturbance of the normal rhythm. The extra systole is simply interposed between two normal systoles. The measuring of the tracing shows that neither of these conditions applies to our case. The extra systole originates, therefore, from the bundle of His.

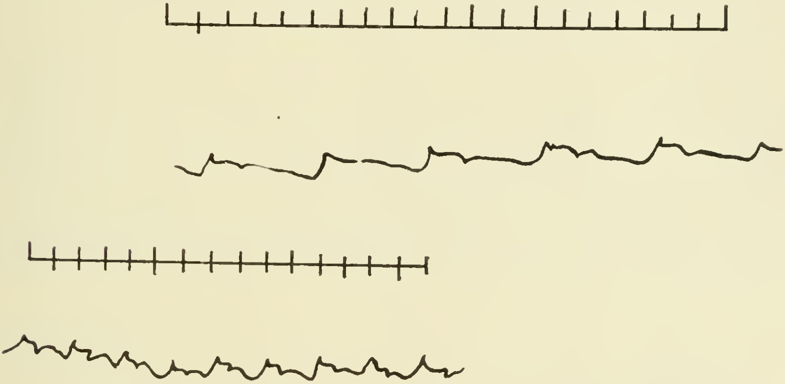

Fig. 5.—The upper tracing gives the carotid pulse during normal heart action, the lower tracing during the attack. Time-marking in fifths of a second.

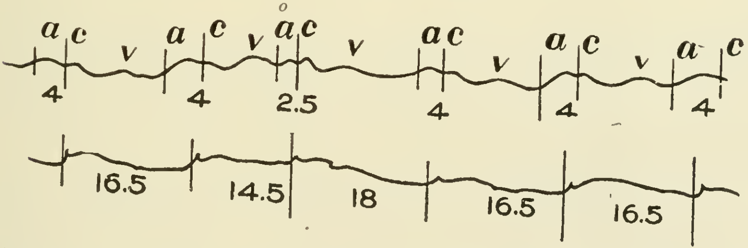

Fig. 6.—The upper tracing represents the venous pulse, the lower the arterial. At the point marked *o* an extra systole occurs characterized by a shortened *a-c* interval, proving its origin from the atrioventricular bundle.

This view is corroborated by extra systoles observed at other times. In these an auricular contraction occurs preceding the extra-systolic beat, and if one measures the distance *a–c*, one finds that this interval is very much shortened (Fig. 6). This shortening of the interval has been shown by Hering[14] to be characteristic of the extra systoles originating from the atrioventricular bundle. An extra stimulus originating from this point progresses to the auricle on one side, to the ventricle on the other. According to the exact location of the place producing the stimulus, both cavities will either contract at the same time or the auricle will contract after the ventricle, or the auricular contraction will precede the ventricular extra systole, but in a shortened interval. In the reproduced tracings we find two of these instances, the simultaneous contraction and the auricular preceding the ventricular in a shortened interval. I have not been able to find any sure instance of the auricular wave following the ventricular.

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[12] Ztschr. f. exper. Path., vol. ii.

[13] Arch. f. klin. Med., vol. lxxxi.

[14] Ztschr. f. exper. Path., vol. ii.

Because of the origin of the extra systole in the His bundle, we find in most tracings the diastole following the extra systole, prolonged, but not to the full length, after which the next normal systole would have followed. In some instances the prolongation is minimal, so that the next systole follows in nearly the same interval as if instead of the extra systole a normal systole would have preceded. In this instance the extra systole is simply the starting point of a new series of normal systoles. The conservation of the normal rhythm after the extra systole would point, according to the experiments of Engelmann,[15] to the venous sinus as the cause of the extra contraction. The study of the venous pulse, however, points unmistakably to the atrioventricular bundle as the point where the extra systoles originate.

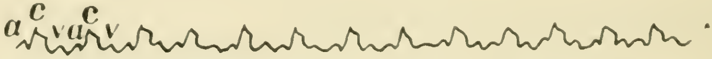

Fig. 7.—The venous pulse during the attack, with the typical three waves: *a*, the auricular; *c*, the transmitted carotid; *v*, occurring at the closure of the semilunar valves.

The form of paroxysmal tachycardia with auricular venous pulse was represented by my case of Dercum's disease. During the period of heart rhythm 140 to 180, the pulse was absolutely regular, and consisted of the three usual waves of the venous pulse—*a*, *c*, and *v* (Fig. 7).

In many instances a change occurs while the heart beats in the rapid rhythm; each second pulse beat becomes very small, to be followed again by a very strong pulse beat. The rhythm of the heart is not disturbed by this allorrhythmia, which represents the typical pulsus alternans first described by Traube, who differentiated it from the bigeminal rhythm by the fact that the intermission following the feeble beat is prolonged in the bigeminal rhythm and normal in the alternating (Fig. 8).

The alternating pulse has been considered by Wenckebach[16] as characteristic of a lesion of contractility. His view was based on the experimental work of Engelmann and Hoffmann.[17] The measure of contractility is given by the height of the contraction, as at each systole the heart contracts with a maximum contraction. The contractility is destroyed by each contraction and recovers slowly during diastole. After a short diastole the contractility does not fully return and a feeble systole results, while a long diastole permits a strong contraction. If one reduces, experimentally, the contractility of the heart muscle, either by causing a frequent heart action (Engelmann) or by stimulating the vagus (Hoffmann), a typical alternation occurs, while the duration of the systole decreases. The cause of the alternation has been found to depend on the fact that the normal heart stimulus is never absolutely rhythmical; it may occur too early in diastole and a feeble contraction ensues. This systole, being feeble, will last a shorter time than a normal systole; the next diastole, therefore, is lengthened, causing a strong systole, which, however, will again lead to a shortening of the diastole. A continuous alternation will be the result.

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[15] Pflüger's Archiv, vol. IV.

[16] Arhythmie, Leipzig, 1903.

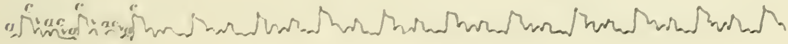

Fig. 8.—A typical alternating pulse.

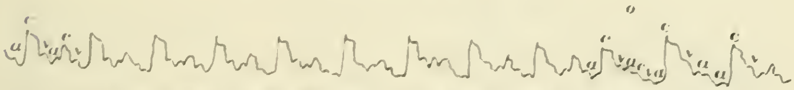

Fig. 9.—An alternating pulse, showing to the right (point o), a systole occurring too early and leading to a disturbance of the alternation.

Fig. 8 illustrates these changes (1 second being equal to 17.5 mm.). The *a* waves occur rhythmically, as well as the *c* waves, at an interval of 0.35 second; the systole in the strong beat lasts 0.17 second; the diastole, 0.18; in the weak beat the duration of the systole is 0.12 second; of the diastole, 0.23.

In Fig. 9 is another instance of the alternating pulse (1 second = 20 mm.). *c* and *a* waves are again rhythmical, each heart evolution requiring 0.3 second. The systole of the strong beat requires 0.175 second, the diastole 0.125 second; the systole of the weak beat lasts 0.1 second, the diastole 0.2 second. At the place marked *o* the pulse beat occurs about 0.25 too early, the following systole is, therefore, extremely feeble, the preceding diastole having been shortened. A very strong systole follows with the effect that the next diastole is so shortened that contractility does not recover sufficiently. The ventricle, therefore, is unable to open the aortic valve, and only the auricular wave appears in the pulse tracing.

[17] Pflüger's Archiv, vol. lxxxiv.

In both tracings an alternation in the auricular action may be noted. The strong auricular beat, however, corresponds to the weak ventricular, and the weak auricular to the strong ventricular. In other tracings not published here the alternation occurs in the auricle and ventricle at the same time. Similar tracings showing an alternation in the auricular action have been published by Volhard,[15] who states that in alternating pulse the small wave in the pulse tracing is of shorter duration than the strong one, for the following reasons: The weakly contracting ventricle will need a longer time to overcome the diastolic aortic pressure; the closure time is prolonged; besides this, the weak pulse wave progresses more slowly in the arterial system than the normal (Extraverspatung). Several tracings in which, with a rhythmical heart action, the duration of the weaker pulse wave in the radical is shorter than that of the strong pulse wave, corroborates his view. That this, however, does not apply to all the cases is clearly shown by Figs. 8 and 9; whereas the retardation of the auricular, as well as the ventricular, wave is clearly shown in Fig. 10

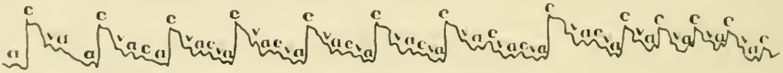

Fig. 10.—The transition from an alternating to a regular rapid pulse.

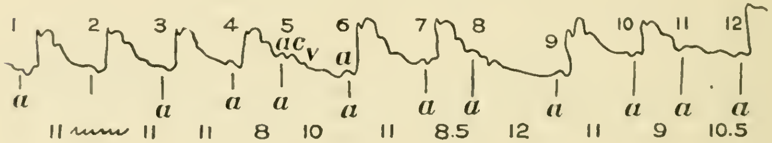

Fig. 11.—The occurrence of extra systoles.

The recovery of the heart from a state of diminution of contractility is shown in Fig. 10. In the beginning the strength of the heart is so diminished that the ventricle is unable in each second beat to open the arterial valves, and only an auricular wave appears; with a partial recovery an alternation sets in, which finally goes over into a regular rapid pulse.

At other times we meet with periods of irregularity which cannot be explained by lesions of contractility. A typical example is given in Fig. 11, showing the jugular pulse. At the beginning the pulse rhythm is regular, about 100 to the minute, each systole lasting 0.6 second. After four pulses an irregularity occurs, showing a premature contraction consisting of three small waves. The first of the waves is undoubtedly the auricular wave, followed by wave *c* and *v*. The interval between the *a* waves measured in millimeters (3.5 mm. = 0.2 sec.) is the following:

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[15] Münch. med. Woch., 1905.

A1–A2—11.0 mm.
A2–A3—11.0 mm.
A3–A4—11.0 mm.
A4–A5— 8.0 mm.
A5–A6—11.0 mm.
A6–A7—11.5 mm.
A 7–A 8— 8.0 mm.
A 8–A 9—11.5 mm.
A 9–A10—11.5 mm.
A10–A11— 8.0 mm.
A11–A12—11.5 mm.

Thus, in three periods the auricular systole occurs too early in the cardiac cycle; an abortive systole ensues, as the contractility has not sufficiently recovered during the shortened diastole. This abortive systole is followed by a regular systole in the normal interval. At times the extra systole occurred so early in the diastole that the ventricle was unable to contract sufficiently to open the aortic valve. Only an auricular wave appeared in the pulse tracing (Fig. 12). Evidence that the ventricle contracted could not be obtained graphically, as no apex tracing could be secured, but it was shown clearly by a booming sound heard over the heart just after this auricular wave. The character of the sound was typical of its extra-systolic origin and differed entirely from the feeble auricular sounds heard in heart block.

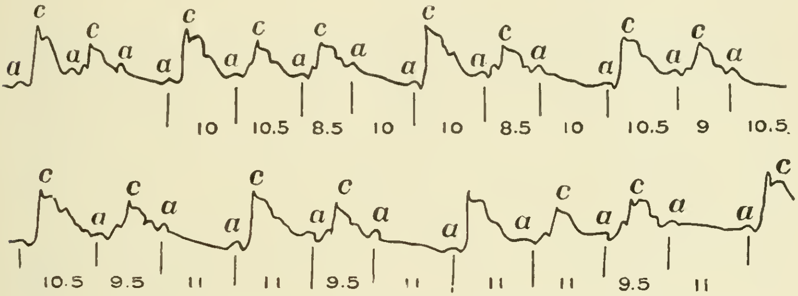
a c a c a a c a c a c a a c a c a a c a c a
10 10.5 8.5 10 10 8.5 10 10.5 9 10.5
c a c a a c a c a a c a a c a c a a c
10.5 9.5 11 11 9.5 11 11 11 9.5 11

FIG. 12.—The occurrence of extra systoles, in which the contraction was insufficient to open the aortic valve; only an auricular wave shows, therefore, in the tracing.

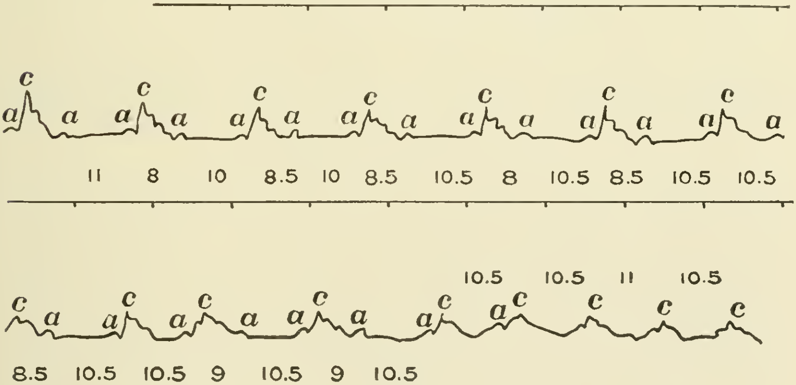

FIG. 13.—The regular occurrence of extra systoles simulating heart-block.

In some instances these extra systoles without ventricular waves occurred regularly, giving the impression of heart block (Fig. 13).

One can easily see that the time between an extra systolic auricular wave *a* and the next auricular wave is equal to the normal time of one heart evolution shown at the end of the tracing—thus proving the extra-systolic character of these contractions.

The similarity to heart block due to a lesion of a conductivity is increased by the influence which the extra systole has on the interval *a-c*, which we are accustomed to consider as a measure of the conductivity (Wenckebach, Mackenzie). During the normal rhythm of the heart the interval *a-c* is equal to 0.2 seconds (Fig. 14, 4 mm.; 2, 0.2 sec.). In the extra systole the interval *a–c* is only 0.05 second; in the following systole 0.1; the normal interval of 0.2 is only obtained in the second following systole.

The shortening of the interval *a–c* in the extra systole points, as in our first case, to the atrioventricular bundle as the place whence the extra systole starts. The shortening in the next following systole can be explained by the rest during the prolonged diastole, so that the conductivity is restored during this contraction. The only point which speaks against the origin from the His bundle is the absence of any compensating interval after the extra systole, a fact which points to the venus sinus as the starting point for the extra stimulus. The study of the tracings of our first case, in which the stimulus proceeded from the His bundle, shows, however, conclusively that in such an extra systole the interval after the extra beat is not necessarily prolonged (Fig. 3). This, in conjunction with the shortening of the *a-c* interval, seems to prove that in this case, too, the tachycardia is caused by extra systoles starting from the atrioventricular bundle. The changing of the rhythm to a tachycardiac attack is clearly shown in Fig. 15. During the slow rhythm the auricular waves occur during the diastole between ventricular beats, occurring too early, and, therefore, being extra systoles. At this point an extra systole occurs very much too early, but is followed by two very small waves, *c* and *v*. The next systole follows at an even shorter interval, again producing a ventricular systole. The rhythm then becomes very rapid, the ventricle responding to each stimulus. The changing of the rhythm

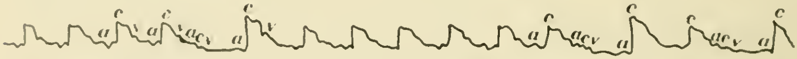

FIG. 14.—Shortening of the *a-c* interval during the extra systole, and in the following systole.

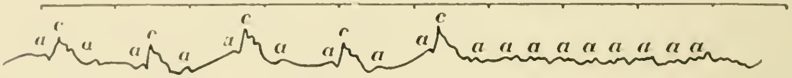

FIG. 15.—Transition from a slow rhythm with interposed extra systoles to a regular rapid rhythm. (Time marking in seconds.)

is clearly due to a series of extra systoles. The regularity of their occurrence does not speak against their extra-systolic nature, as we have seen the regular occurrence of extra systoles in Fig. 8. Similar tracings with rhythmical extra systoles have been published by Gerhardt and Lommel.[19]

Tracings showing the pathogenesis of the attacks were only obtained in these 2 cases, but in other cases auscultatory evidence of the extra-systolic character of the paroxysmal attack could be obtained. It would be entirely hypothetical to try to explain the attack from a physiological standpoint. Whether disease of the vagus nerve alone or in combination with an irritation of the accelerans may cause such an attack is difficult to determine in the absence of physiological experiments. Further investigations may clear up the question, in which conditions the point of origin for the stimulus changes from the venous sinus to the atrioventricular bundle.

The argument presented in this paper, however, emphasizes the pathological importance of the atrioventricular conducting system. It is interesting to note that paroxysmal and permanent bradycardia, as well as paroxysmal tachycardia, can be referred to a lesion of the His bundle.

DILATATION OF THE HEART.[1]

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It is not my intention to go into a detailed discussion of dilatation of the heart, but only to touch upon some of the more important points, more especially the etiology and treatment.

Dilatation of the heart is associated with either a thickening or a thinning of its walls, more commonly the former, and may be looked upon as an index of the disproportion between the intracardiac pressure and the resisting power of those walls. One or all of the cavities may be affected.

Etiology. The majority of instances of cardiac dilatation may be said to result from one of the following factors, operating either singly or collectively: (1) An increase in the amount of work to be done by the heart, as in general arteriosclerosis, chronic nephritis, valvular disease, overstrain, etc; (2) an impairment of the power of the myocardium from degenerative changes, toxic conditions, etc.; and (3) failure of the cardiac muscular tone, as seen in some instances of severe nervous shock, etc. Thus we see in arteriosclerosis and in chronic nephritis, hypertrophy and dilatation of the heart due to increased demands upon this organ. In valvular lesions of all varieties there is almost sure to occur more or less dilatation, usually with hypertrophy. Examples of overstrain are to be seen in athletes and in those whose occupations require great muscular effort. Oarsmen and long-distance runners necessarily tax their hearts to the utmost. The cardiac pulsations are greatly increased in force and in frequency to supply the increased demands of the muscular system and to accommodate the rapid return of the blood to the heart, but this may not be sufficient. Besides, the mechanical overfilling of the cardiac chambers, which produces a severe strain on the contractility of the cardiac muscle, there are also the fatigue products which probably have a toxic effect on the muscle fibers. With this combination at work, there is small wonder that the heart gives way under the strain, and dilates in some instances. I am aware, however, that there are some observers who do not believe that a sound heart ever dilates from excessive muscular exertion alone.

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[19] Arch. f. klin. Med., vol. lxxxii.

[1] Read at a meeting of the American Climatological Association, Washington, D. C., May 7, 8, and 9, 1907.

Among the more important causes of myocardial degeneration are the acute infectious diseases, such as diphtheria, influenza, and rheumatism, if we may class the latter as such. According to Lees[2] myocarditis is far more common in rheumatism than in diphtheria and influenza, but much less dangerous, and the microscope shows much less destruction of the muscle fibers. Not a few deaths result from acute dilatation in diphtheria or shortly after apparent recovery, and many instances of weak heart finally presenting the picture of dilatation date back to an attack of epidemic influenza. A number of writers have called attention to dilatation in typhoid fever, notably Louis, Andral, Gunsburg, Wunderlich, and Stokes. The condition may be general or noted on either side of the heart. Romberg[3] found considerable dilatation of the right ventricle and right auricle with a contracted and hard left heart. A typical example of cardiac dilatation in a case of typhoid fever came under my observation last fall, developing in the fifth or sixth week of the disease and rapidly ending fatally.

In the more intense forms of anemia, cardiac dilatation is the rule, and probably accounts for many of the murmurs which we are wont to call hemic. Myocardial degeneration seen in those past middle life is most commonly due to failure of the blood supply of the cardiac muscle, resulting from sclerosis of the coronaries.

After considering the many definite and tangible causes of dilatation, such as myocardial degeneration, valvular lesions, nephritis, etc., there still remains a fairly large class in which we are not able to find perfectly satisfactory explanation. Here we must fall back on the nervous system and say that the cause is loss of muscular tone; indeed, this seems not by any means an improbable solution—in fact it is urged by Colbeck[4] that all forms of cardiac dilatation may be directly traced to loss of muscular tone. By muscular tone we mean that state of tension in which the muscles are held—which is dependent on the nerve supply.

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[2] Brit. Med. Jour., 1901.

[3] Erkrank. d. Herzmuskels bei Scharlach., Diph., u. Typhus, Arch. f. klin. Med., xlvii.

It is a well-known fact that severe shocks, joy, grief, or any severe nervous strain may cause a dilatation of the heart, and, as Broadbent has said, death may result actually from a broken heart. Immediate death under such circumstances, however, is prabobly due to cardiac inhibition. I am inclined to believe that small doses of opium, which often seem to act so satisfactorily in dilatation, produce results largely through the soothing effect upon the nervous system. Cheimisse[5] states that Marmorstein reported two instances of cardiac dilatation from shock during the rioting in Odessa. The patients were neurotic, and the sight of murder and pillage was the immediate cause. Many such examples are to be found in literature.

It is quite conceivable that under prolonged severe exertion even the perfectly normal heart might, and we know does, become exhausted and loses temporarily its muscular tone. In the majority of instances this form of dilatation is rather quickly recovered from, but not always. It is a well-known fact that the training which an athlete goes through for a strenuous competition is to give the muscles, and the heart muscle in particular, a chance to hypertrophy. The young, from their mode of life, are always more or less in training; hence the violent strains which their hearts are called upon to bear, do no specific harm, except, I believe, in rare instances; but it is a vastly different matter when a man past forty, who has given up active exercise for some years, is called upon for some violent effort, such as a sprint of a couple of squares to catch a car, or who, because of a too irascible temper, becomes involved in a bodily conflict. If this happens to one whose heart is under the influence of nicotine or some such toxic agent, or who has a pre-existing heart trouble, such as a valvular lesion or a myocardial degeneration, dilatation of a serious nature may quickly develop with all the usual indications.

The exciting cause may be any unusual muscular or mental effort. I recently saw a prominent lawyer who, though well advanced in years, had always been a man of remarkable vigor, who brought on the symptoms of dilatation by the preparation of a long and important brief. He had just concluded his arduous task when he was suddenly seized with dyspnœa and oppression in the cardiac region. I found the heart considerably enlarged, acting very irregularly, and a mitral systotic murmur present. Whether this murmur was due to a true valvular lesion or to a relative insufficiency from dilatation I am unable to say, as he left for his home in the West as soon as he had partially recovered.

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[4] Lancet, 1904.

[5] Semaine méd., 1907, No. 9.

Another gentleman, whom I had the opportunity to observe for a number of years, suffered a severe dilatation from over-fatigue of body and mind during an active political campaign, and a second very acute attack, which nearly cost him his life, was the immediate result of rather vigorous abdominal massage. In this case there had been for many years completely compensated mitral and aortic insufficiencies, with but little enlargement of the heart.

Again, the symptoms of dilatation may be brought on by an acute illness in those who, though the subjects of valvular lesions or some other disorder of the heart, have acquired perfect compensation. I had such an instance under observation while writing this paper. A woman aged forty years, who has for a long time shown the physical signs of a mitral stenosis, developed a bronchopneumonia, with a very harassing cough. After a few days of constant coughing, marked orthopnœa, with œdema of the lungs, developed. Examination of the heart showed a loud systolic murmur in the tricuspid area, in addition to the pre-existing mitral stenotic bruit. Caffeine in liberal doses gave fairly prompt relief, and the wall of the right ventricle so far regained its tone that the systolic murmur was scarcely audible at the last examination.

One of the more recent instances which has come under my observation was in the case of an old lady who suffered from a myocardial degeneration. One night she got out of bed, slipped on the floor, and, in a violent struggle to regain her feet before anyone should find her up alone, brought on a marked dilatation, from which she almost died. It is useless to multiply examples of this well-known condition.

SYMPTOMS. Sleeplessness is not infrequently the first symptom to attract the attention of the patient. Sometimes it is referred to the violence of the heart's action or to an uncomfortable feeling of nervousness, or to a cough, evidently due to faulty circulation. In other instances there is actual dyspnœa or even orthopnœa. It is a strange fact that these sufferers often rest quietly for some hours after retiring, only to be awakened in the early morning by one of the above-mentioned symptoms.

The possibility of cardiac insufficiency should always be borne in mind when insomnia develops. A stimulant in such instances is often the best hypnotic. I well remember one patient in whom a small dose of digitalis produced such prompt sleep that it was mistaken for an opiate. There is usually complaint of weight and oppression over the precordium—seldom actual pain. Sometimes the dilatation seems to make itself actually felt, the patient saying that he feels as though the heart were too big for the chest.

The pulse is usually rapid and, not uncommonly, arrhythmic. Dyspnœa at first only on exertion, later becomes more or less constant. Cyanosis and œdema of the ungs may develop, the latter being one of the earliest and most persistent symptoms, as evidenced by cough with seromucous expectoration, which may be slightly pinkish or quite bloody, depending on the amount of congestion. In acute dilataton œdema of the lungs is one of the most urgent symptoms and not infrequently appears to be the immediate cause of death. General œdema is usually of later development, when there is no longer any doubt as to the diagnosis. Many of those whose hearts are undergoing gradual dilatation suffer chiefly from gastro-intestinal disorders—flatulent dyspepsia or nausea, due to congestion of the abdominal organs; and it is often difficult to convince them that the heart is the cause of their trouble. The liver becomes congested and often greatly enlarged and painful to pressure, even if there be no complaint of heaviness in the right hypochondrium. Congestion of the kidneys results in scanty, more or less albuminous urine, in which casts are usually to be found.

Diagnosis. The diagnosis is based upon the presence of the above-named symptoms and upon the following physical signs: The whole precordium may seem to throb with each systole of the ventricles, when there is an associated hypertrophy of much magnitude; while in instances of dilatation with thinning of the walls there may be no visible impulse whatever, or only the merest undulatory wave in the neighborhood of the apex. The jugular veins may be distended and may pulsate. If the right ventricle is dilated, the epigastrium may pulsate. The apex beat is nearly always displaced to the left, and downward if the left ventricle is involved. By laying the ulnar surface of the hand parallel to and to the right of the sternum, a fair idea of the extension of the heart to the right may be obtained by the impulse which is often palpable to this sensitive part of the hand, although one is likely to get a slightly exaggerated impression of the size of the heart by this method. My attention was first called to this valuable point by C. H. Hoover. In percussion of the heart, I wish to lay great stress upon the advantage of the palpatory method over all others; its accuracy is often very gratifying. The cardiac dulness will be found to be increased to a greater or less degree, depending on the dilatation. An accurate determination of the area of dulness is of the greatest importance, and at the same time requires more skill and practice than any other part of the examination There may or may not be heard endocardial murmurs, due to true lesions of the valves, but there is likely to be present the systolic murmur of a relative insufficiency of the mitral or of the tricuspid valve or of both. Time alone will prove this point. The disappearance of the murmur of a relative insufficiency is always encouraging, as it is an indication that the cardiac chambers are returning to the normal size.

The sphygmomanometer has been rather a disappointment as an aid to diagnosis in diseases of the heart. It is of value, however, in giving warning of possible danger ahead. For instance, the discovery of an abnormally high blood pressure at once suggests the presence of conditions, such as arteriosclerosis or chronic nephritis, which will sooner or later be likely to produce hypertrophy with dilatation, and we are able to direct the life of the patient accordingly. The use of this exact method of estimating the pressure also makes one much more accurate in the examination of the superficial arteries in determining the real force of the pulse. A point which was at first a surprise to me was the high blood pressure so frequently observed in spite of the very evident symptoms of cardiac insufficiency. I mean especially in the first few days of what might be called an acute dilatation, when there was dyspnœa, œdema of the lungs, arrhythmia of the pulse and other indications of circulatory insufficiency. As the condition improved the pressure fell, and the reduction occurred in spite of the constant administration of digitalis and cardiac stimulants other than the nitrites. These observations were upon cases of dilatation with hypertrophy. The elevation of pressure under such circumstances would appear to be due to exaggerated efforts of the heart and vessels to reestablish the circulatory equilibrium.

Differential Diagnosis. The two conditions with which dilatation might be confounded are simple hypertrophy and pericardial effusion. The former requires but little consideration as the symptoms are not the same, unless the walls of the heart have begun to relax, when it falls into the class now under discussion. A pericardial effusion, however, is by no means so easily differentiated from dilatation, as the symptoms may be practically the same. In the former the cardiac impulse is generally less distinct, the first and second sounds weaker, and most important of all—there may be heard a pericardial friction rub, even though there exists a large effusion. It must be borne in mind, however, that the two conditions may co-exist. In effusion the area of dulness is of the classical irregular dome-shape, the apex being upward, and extends lower down, depressing the left lobe of the liver in some instances. The relation of the extent of dulness to the position of the apex beat, when it can be felt, is of importance. In effusion the dulness extends to the left and below the position of the apex beat. Dulness in the right fifth interspace, as pointed out by Rotch, is a valuable sign of effusion, as is also the broadening of the area of dulness toward the base of the dome when the patient assumes the sitting posture. If there still be doubt, the question may be quickly settled by the use of the fluoroscope, when the apparatus is available; the dilated heart will be seen to pulsate while the effusion will remain stationary.

TREATMENT. In an endeavor to relieve this conditon it is necessary to bear in mind the two cardinal objects: To lessen the amount of work which is required of the heart, and to increase the power of the heart to do its work. The first may be accomplished by absolute bodily and mental rest, by a light diet, by active purgation, by relaxing the peripheral vessels, and by bleeding. The second may be accomplished by the administration of cardiac stimulants, by baths, by judicious exercises, etc.

Prophylaxis is worth much in cases of beginning dilatation. If these patients can be got under control when the first indications manifest themselves, many attacks may be warded off by advice as to overstrain, either mental or physical. If men with large interests and women with excessive household or social cares can be induced to lighten their loads of responsibility; if heavy eaters can have their appetites controlled, much may be accomplished without recourse to medicine.

Even after serious symptoms have actually developed, much may be done without cardiac stimulants. Some of our patients yield promptly to rest in bed, a dose of calomel or blue mass, followed by salines. If this fails to restore the circulatory equilibrium, digitalis is turned to by the vast majority, and I, too, usually give it the preference; but caffeine is, in my opinion, a very close second and not infrequently is superior. I think that I have noted this superiority especially in cases of high blood pressure, which would be easily explained by the difference in physiological action of the two drugs. But at this point I want to emphasize the fact that digitalis in some cases is indispensable, in spite of high blood pressure, in spite of the most marked calcareous degeneration of the arteries, as far as they can be felt. In some instances our best efforts along these lines will fail, and the symptoms even grow worse—cyanosis and dyspnœa increase, nausea develop with extreme restlessness, and the heart's action become more irregular and rapid. These are the cases which require more heroic handling, and these are the cases, if properly selected, which are often benefited by bleeding. The patients who are best adapted to blood-letting are the plethoric—more usually men who, though suffering from some heart lesion, have, through careful dietetic and hygienic regimen, been made to increase in weight and strength, and who, by some indiscretion, have been smitten in the midst of comparative health.

Some prefer wet cups or leeches, and they may be very beneficial. Incidentally, I might say that I cannot see why their application over the precordium, in preference to any other area, should be of especial advantage, unless they should also act as counterirritants. My own experience has been with venesection; hence, I prefer the very simple method of opening a vein in the forearm and drawing off from 8 to 16 ounces of blood. The results are in some instances most gratifying. I have seen the cyanosis improve at once, the nausea disappear while the blood was still flowing, and the patient express himself as much more comfortable. This method of relieving the over-distended heart appeals to me as being one of the most rational at our command, and yet it is surprising how few ever resort to it. By unloading the venous circulation, the overfilled heart is enabled more completely to empty itself, and the overstretched muscle fibers are once more allowed an opportunity to contract to somewhere their normal dimensions. After bleeding, cardiac tonics produce results which previously seemed unattainable.

I shall not mention the names of the army of drugs which have been used in dilatation of the heart, but will content myself with the mention of one more, which, if properly given, will be found most useful—that is opium. These patients are often possessed with that spirit of unrest, that condition of general wretchedness which it is hard to describe, but which is perfectly familiar to us all. For this symptom-complex there is nothing so useful as small doses of morphine. I have found as little as $\frac{1}{32}$ grain every four hours most beneficial; not only does it quiet the nervous system, but it seems to stimulate the heart, and in most instances appears to do no harm. I shall not take up the treatment of dilatation by exercises and baths. A discussion such as the present would hardly be complete without some mention of the most acute forms of dilatation. In these the anxious, terror-stricken expression, livid skin, thready pulse, with cold and clammy sweat, indicates that death is imminent. The free use of nitroglycerin has, in some instances, seemed to be life-saving, and I have rather learned to rely upon it under such circumstances. Caffeine is especially useful when œdema of the lungs is pronounced. In fact nitroglycerin, caffeine, and digitalis may be given at the same time, as their promptness of action is about in the order named, so that the effect of the first two will have entirely disappeared before the third makes itself felt. Nitroglycerin should, in my opinion, be reserved for emergencies such as the above and for the attacks of great oppression which are likely to come on from time to time in the less acute cases of dilatation. Its continued use is depressing. I have not mentioned strychnine because I have rather lost faith in it, having observed better effects from other remedies.

Diet. Upon this point there are two widely different opinions. The one, that only liquids should be given; the other, that a dry diet is preferable, on the ground that the more liquid is taken into the system the more will be taken up by the vessels, and consequently the greater amount of fluid will have to be moved by the already overladen heart. After trying both methods, I have concluded that the dry diet is to be preferred, unless there are decided uremic symptoms to be considered. Especially is this true when there is, as is so often the case, a tendency to tympanites.

A RESUME OF THE PHYSIOLOGY OF THE KIDNEY, WITH A VIEW TO THERAPEUTICS.[1]

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A review and, as far as possible, a fair summary of the state of our knowledge of the physiology of the kidney seems to me to be quite worth while, especially in view of its great importance to those who are combating disease; because of the very rapid advances and the many changes which our knowledge of the functions of the kidney, synthetic, analytic, and secretory, have recently undergone; and because the old-time theories proposed by Ludwig and Heidenhain are, with this increase in knowledge, being gradually modified by their respective followers, and in many important points approaching each other; though it must be admitted that the theory of Ludwig, as was to be expected from its inherent simplicity of explanation, is being widely deviated from. I will discuss at the close of the paper the probable constructive lines for a new theory. First, I wish to adduce the important facts.

I. The Internal Secretion. The chief arguments in favor of this theory suggested by Brown-Séquard are as follows:

1. It has been observed in both men and animals that occlusion of both ureters leads but slowly to death (men have lived frequently fourteen to sixteen days), and that death ensues in man (it is harder to judge of this in animals) without marked symptoms of uremia, often with hardly a symptom that could be classed as uremia. In Fowler's summary of 93 cases, more than 66 per cent. showed no marked uremic symptoms. On the other hand, in animals and in man, when accidentally a single functional kidney has been removed, death occurs more rapidly (four to six days in man), though at least 1 case is on record in which a man lived eleven days; nor do all cases show marked uremic symptoms, though they are more common than in the first group. There is, however, it must be noted, a very marked difference in the severity of the operations for ureter closure and kidney removal in animals, and in man the ureters have rarely been closed by operative interference, and then only in severe operations. This fact must be seriously considered when considering the value of the differences between the two groups of cases.

2. Bradford and others have noted very marked wasting of the body and increased loss of nitrogen accompanying the polyuria which supervenes when five-sixths of the kidneys are removed. This, too, may be ascribed to an internal secretion.

3. The pharmacological experiments of Brown-Séquard and

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[1] Read at a meeting of the American Therapeutic Association, Washington, D. C., May 7, 8, and 9, 1907.

others who tried to reduce an impending or existing uremia produced in animals by double nephrectomy by injections of (*a*) glycerin extract of kidney, or (*b*) defibrinated blood from normal animals, taken either from kidney veins or the general circulation. Brown-Séquard himself was the first to experiment with the glycerin extract. His experiments seem to have been few in number and are not reported in detail. He himself subsequently appears to place more reliance on his pupils' work and upon general considerations such as the above. Ajello and Parascandalo operated on twelve dogs who died in from four to forty-eight hours; of ten other dogs which, after similar operation received glycerin extract injections, one lived four days, six others three days, the other three died in from forty-eight to fifty-two hours. This is not very convincing, especially when the controls died so early—four hours even; much longer survivals than forty-eight hours have occurred in similar experiments reported by other observers. Their rabbit experiments are not more convincing. The work of Meyer, a pupil of Brown-Séquard, whose work is considered by the latter as conclusive, s still less satisfactory. In one series of experiments he chose the cessation of dyspnœa, as recorded graphically, as a sign of improvement. Neglecting the fact that the method of recording might lead to deception, the criticism used is hardly a satisfactory one, as is urged with reason by Tigerstedt and Lewandowsky. His experiments are only five in number—three dogs and two rabbits. He used for injection blood of normal animals. Meyer, and also Vitzou, carried on a series of experiments in which the length of postoperative life with and without injections of blood was compared. Animals receiving injections had certainly longer postoperative lives, but that this was due to an internal secretion is rendered doubtful by the experiments of Chatin and Quinard, who showed the blood serum, which one would expect to contain the secretion, had absolutely no effect in prolonging the animals' lives. They have a longer and more complete experimental series than have either of the others. It seems that either the internal secretion must be carried by the corpuscles or that the corpuscles aid in prolonging the postoperative lives, if, indeed, they were prolonged in the experiments reported above.

4. Therapeutic experiments with extracts of kidney on uremic and nephritic persons are reported as successful by Dieulafoy, Tiessier and Frankel, Jacquet, Concetti, and Chiperowitz. A careful analysis of such cases as I have been able to see are not convining. Comparatively sudden changes for the better are not rare in nephritis. The treatment seems to have fallen into disuse since, in the literature available, I have found no reference to it since 1898.

5. As shown by several observers, uremia has supervened in nephritis without evident nitrogen retention. This is an unexplained fact.

I think it unnecessary to consider the experiments of Tigerstedt and Bergmann, so completely nullified by those of Lewanowski, nor the evidence that some have tried to deduce from the variations in hemolytic and toxic properties of serum and urine in cases of nephritis.

To sum up, I think that Brown-Séquard's suggestion has been rendered not more, but rather less, plausible than when first communicated. Great loss of functional kidney substance does lead to an abnormal metabolism, but this by no means proves an internal secretion.

II. SYNTHETIC AND ANALYTICAL FUNCTIONS. The mention of the paper of Barcroft and Brodie, who showed that increase in the excretory function of the kidney led to an increased consumption of oxygen and increased giving off of carbon dioxide in the blood, leads me to speak of the other known chemical activities of the kidney.

1. *Synthetic*. I need but to recall that the kidney can synthetize hippuric acid from glycerin and benzoic acid, salicyluric acid from salicylic acid and glycine. Also carbonic acids of many other nuclei in addition to that of the benzene nucleus, such as, furan, thiophene, and pyridin can be excreted in similar combinations. That this is a function of the kidney and doubtless of the tubules, has been still further confirmed by the success of Abelous and Ribaut in isolating to a certain extent the ferment responsible therefor.

2. *Analytic*. Schittenhelm has distinctly shown that the kidney contains a ferment which is able to split uric acid into urea, and also ferments which desamidize and oxidize other purin bodies. It is possible also that kreatinin, which, as Landois showed, is toxic with symptoms resembling uremia, may be formed by dehydration in the kidney from the nontoxic kreatin. Doubtless other synthetic and analytic functions will be discovered, and it may well be that their disturbance will, in large part, explain the causation of uremia.

III. TUBULAR EXCRETIONS. Nevertheless, these functions are, on the whole, subordinate to the main function of the excretion of urine.

1. The paper of Ebstein and Nicolaier, and, more convincingly, those of Sauer and of Anten, show that uric acid is excreted by the cells of the convoluted tubules and also, possibly, by those of the ascending loop of Henle. Undoubtedly those cells which carry on synthetic and analytic activities will also cast their products into the tubular lumen, and we will not be far astray if we localize these functions also in the tubule. Some experiments performed by Tropp show that such a synthesis as that of hippuric acid is not accompanied by any increase in water excretion. I have lately tried in three experiments to find an increased flow of urine when uric acid is secreted in large quantities, but as yet all experiments have been completely negative. Greatly increased uric acid excre-

tion was accompanied by a decrease, not an increase, in water. Phloridzin is known to be a poison which attacks the tubules, and some evidence exists that the passage of dextrose into the urine after the use of this poison is accompanied by loss of water. This may also be true of such poisons as cantharidin and possibly calomel. The pigments are also probably excreted by the cells of the convoluted tubules, as injected foreign pigments have been shown to be by Hober and Königsberg.

Attention must also be called to the fact that the urine leaving Bowman's capsule is alkaline, as shown by Dresser, while in the convoluted tubules it becomes acid. This is probably due to an increase in the free hydrogen ions, owing to the passage of CO_2 which, in the experiment of Barcroft and Brodie, does not appear in the venous blood in proportion to the oxygen lost by the arterial blood.

2. The recent papers of Bainbridge and Beddard and of Cullis and Brodie, if confirmed, as is probable they will be, will prove that the tubules in the frog, at all events, can excrete water, urea, chlorides, sulphates, probably kreatinin and phosphates, and dextrose when there is hyperglycemia or when poisoned with phloridzin. Caffeine sodium benzoate also seems to stimulate to excretion (note this body contains two substances which should influence tubules). It is, of course, doubtful how far we can apply to man results obtained on frogs in which the relations of the blood supply are so different, the tubules having, as well as the arterial, a secondary venous blood supply from the lower limbs; still, if we do so, the following points in the experiments of Cullis are of importance: the tubules on a venous supply did not excrete except when stimulated; neither phosphates, chlorides, nitrates, nor sulphates acted as such a stimulant; urea was a weak or doubtful stimulant, while phloridzin and caffeine sodium benzoate were the successful stimulants. The work of Loewi and Schmid would also go to show that sugar is excreted by the tubules when poisoned with phloridzin. The increase in urine flow accompanying it they consider to be due to a prevention of water absorption.

Accepting the observations of Cullis and Brodie on frogs, it is apparent that the diuretic salts, sulphates, phosphates, carbonates, if in excess in the body, must be got rid of by the glomeruli. This is the view most generally accepted, though Meyer and others do not fully concur in it. Before, however, we consider the excretion of salts, I wish to draw attention to the fact that under any but the most extreme experimental conditions an excess of such salts cannot occur in the blood without at once introducing osmotic pressure differences between the blood and the tissues, and that these are at once done away with, first, by water passing into the blood; and, second, by salts passing into the tissues. Very rapid salt absorption will lead to hydremia.

3. *Inorganic Salts and Urea.* The following facts are to be noted in regard to urea and nitrogen excretions: Urea occurs in normal urine in higher concentration than in the blood plasma (100 c.c. of urine often contains as much urea as 2 liters of plasma).

The more rapid the flow of urine, in general, the smaller the percentage of urea; the greater the daily quantity, the lower the percentage; but the total daily amount of urea will vary only with the daily nitrogenous katabolism.

Especially in cases of nephritis, the output of urea does not necessarily keep pace with its intake nor with katabolism (heaping up of non-coagulable nitrogenous bodies has been shown to occur in the blood), and this in cases in which water excretion appears normal; in other words, nitrogen retention with normal water excretion. The cause of such retention does not necessarily reside in an inability on the part of the kidney to excrete urea, though it may be so in some cases. Such nitrogen retention may persist for a long time (as in a case reported by Draper, for thirty days, and amount to 146 of nitrogen) without uremic symptoms.

4. *In regard to chlorides:* retention of chlorides in nephritis leads almost at once to a retention of water. Retention of salt has amounted to 40 to 50 grams, and was accompanied by proportionate increase in œdema. So many cases are now on record in which chloride retention has been shown to be the cause of water retention, that Marie has been led to frame the following hypothesis: that at first, on retention occurring the tissues are able to fix and retain a certain amount of chloride; when this is surpassed, water retention must occur. With restored chloride excretion, water also is given off. But the failure to excrete chlorides is rarely or ever absolute; retention occurs only above a certain daily intake. If the salt intake be reduced below this limit chloride elimination occurs, and, with it, water (Widal, Halpern, Claus, Plaut, and Reach). Increase in intake sometimes seems to hinder excretion.

5. *Sulphates and phosphates* may, like the chlorides, vary independently of their intake, and also of the nitrogen output, or temporarily of the water. Such massive retentions as are reported for chlorides, however, do not occur, and, as a whole, their daily amounts vary with the food intake and daily katabolism.

All these salts are present normally in the urine in larger amounts than in the plasma, but the more rapid the urine excretion, the lower their percentage, until it may happen that in extreme cases traces alone of chlorides occur. The urine may also come to have a higher freezing point than the plasma, that is, contain a much lower salt percentage (Dreser, Brodie, Henderson, Frey, etc.).

The excretion of a urine with a lower salt content than the normal blood for a long time—several hours in animal experiments—as shown by Achard and Paisseau, Reichel, Brandstein, and others, causes definite degenerate changes in the convoluted epithelium.

Leopold has also shown recently (his paper much needs verification) that feeding to rabbits and dogs a not excessive amount of salt daily (rabbits from 0.05 to 1 gram), also led to changes in these same convoluted tubules. He also showed that the changes caused by phosphates were much more marked.

6. *Water*. In regard to the excretion of water even the most ardent advocates of the secretory activities of the tubules suggest the glomerulus as the site of the main water excretion. The phylogenetic development of the kidney suggests it; the glomerulus at first is projected into the body cavity near the inner end of the neophrostome, whose mouth is lined with cilia. It must also, however, be noted that it was then the main, if not the sole, excretory apparatus for other body waste. The histology of the kidney suggests it. The afferent vessel to the glomerulus is larger than the efferent; the efferent vessel breaks up about the lower tubules to form a second capillary network. Hence the blood pressure in the glomerulus must be high; the pressure in its capillaries higher than elsewhere in the body. The epithelium of the glomerulus is thin and very flat, and probably syncytial. It is not gland-like in appearance.

Increased water production is favored by (*a*) an increase in blood flowing through the kidney (Starling, etc.). This must be qualified by the reservation that the blood may not be too concentrated. Observations are on record in which, though the blood flow through the kidney increased when caffeine was given, yet little or no secretion occurred, and it has been shown that in these cases the percentage of water in the tissues was low (Henderson). (*b*) Increase of blood pressure from the arterial side, if not accompanied by a decreased flow through the organ, increases the flow. Impeding venous outflow decreases water excretion. There is a certain minimum blood pressure below which no urine is excreted. This minimum varies with the composition of the blood—the more hydremic the blood, the lower the minimum; it is probably between 30 to 40 mm. Hg. in man (Starling, Henderson). Caffeine has been shown to dilate the vessels of the kidney, and, at the same time, by its heart action the blood pressure does not fall (Loewi, Brodie, etc.). (I believe that only when the kidney vessels are greatly dilated before injection does dilatation not occur.) That means an increased flow of blood through the kidney under equal or increased pressure. It may fail as a diuretic if the patient be poor in water. Other purin bases act as diuretics in the same way. As pointed out above, caffeine may possibly lead to increased activity of the tubules as well. Digitalis is not a diuretic in normal individuals, but is invaluable in those cases in which the circulation is poor. With improvement in the circulation comes diuresis. It may fail, of course, in those œdematous cases in which the œdema is due to chloride retention. Mercury salts, as pointed out above, are poisons for the tubule cells, and no doubt act as such. Hydremia is apt to dilate the vessels of the kidney, but is not constant in its action. (*c*) The presence in the blood of increased amount of salts, with high osmotic equivalents, sulphates, phosphates, carbonates, as well as the occurrence in increased amounts of salts with not such high osmotic equivalents, chlorides, dextrose, nitrates, increase the flow. All these act largely, first, by increasing the water content of the blood; second, by carrying water with them into the tubules and retaining it there (Loewi, etc.); and third, evidence is also to be found in many papers that salts in excess may also cause a local dilatation of the vessels of the kidney (Magnus, Brodie, etc.). (*d*) The addition, by the tubules, of dextrose or other crystalloids increases the flow.

Reversal of the above conditions, decrease in blood flow, in the amount of salt in blood stream, etc., will decrease the flow of urine.

Finally, in nephritis, damage may lead to a primary inability to excrete water, and, on account of osmosis, salts must be retained, and thus greatly decrease the amount of urine.

Having thus briefly, and necessarily imperfectly, but I hope not unjustly, reviewed the facts in regard to kidney function, may I be allowed briefly to sum up the facts as they stand, and contribute in a word or two a picture of the production of urine. First, the cells of the tubules do undoubtedly excrete urates, hippurates, pigments, and, possibly, creatinin. They are permeable, and, in the frog, as in man, may excrete water, chlorides, urea, phosphates, sulphates, under stimulus, and, under still more abnormal circumstances, dextrose (phloridzin hypercemia). Second, the urine leaving the tubules, though usually more concentrated than the plasma in salts and urea, under a rapid flow more closely approximates to it in salt composition, and may even in some cases become much more dilute than the plasma, as in, experiments spoken of above and in diabetes insipidus. The first of these facts, great concentration, may be explained by very great water absorption by the tubules, which, there is evidence to show, does occur. The second, only by very greater absorption of salts by the tubules, and this is especially hard to understand in cases such as diabetes insipidus, in which the body really seems to need water. This necessary supplementary explanation, taken in conjunction with all our more recent knowledge of living animal membranes, many subsidiary difficulties not mentioned in the paper, and facts in regard to the definite excretory functions of the tubule spoken of, so overloads and modifies Ludwig's conception of the excretion of urine as to make it no longer tenable, even if we make, as has been pointed out to be necessary, the modification of understanding for "filtration" "diffusion modified by pressure." Then must we, with Heidenhain, say the glomerulus and tubules secrete. It appears to me to be a matter of definition. Heidenhain, when he said secrete, meant, I think, secretion, such as is carried on by other glands.

Let us take a typical secreting gland, such as the submaxillary, and see what secretion means in its case. It is, first, supplied with nerves, stimulation of which causes a flow of secretion even without increased blood flow. The kidney appears to have none such. Such a gland, on appropriate stimulation, nervous or chemical, secretes, and even against considerable pressure, even if the blood supply be poor; in the extreme case even if it be absent. The kidney in rabbits fails absolutely to secrete if the blood pressure be lower than 30 mm. Hg. and the blood normal. Caffeine is looked upon as the most efficient chemical stimulus we have (salts and water cause changes in the blood and urine which may greatly alter the conditions); yet, as I have shown, when the body is poor in water, caffeine fails as an excitant to secretion, though it brings about the necessary vascular changes. Such a gland, furnished with normal blood, will secrete when stimulated against a pressure many times the blood pressure of its vessels. This the kidney fails to do, although the glomerulus does not alone excrete, and there is no very great distance from the convoluted tubules to the ureter, but here, it must be noted, that the excretion of the tubules is not necessarily accompanied, as shown by Tropp and myself, to be accompanied by much water. Change in renal blood pressure is exactly and instantly followed by the pressure in the ureter, and a fair flow of urine, with a high blood pressure, will give a greater ureter pressure than a faster urine flow with a low blood pressure. We have, therefore, in the kidney a different type of secretion. Why not drop the word, with its associations, and say excretion?

If both these old theories have proved to contain fallacies, is there any theory which can be propounded to explain the production of urine? I believe we shall soon be able to answer in the affirmative. May I be permitted to indicate what I think to be the probable constructive lines. On the one hand, we may believe that the glomerulus excretes water, urea, chlorides, and possibly other salts, and that it must be so delicately adjusted as to respond to very small variations in the amounts of these substances in the blood and have a selective excretory activity for each substance, which may be either increased or decreased by disease, etc. Sulphates and phosphates may be added by the tubules with such other substances as the urates. It may even be that each constituent is added by a different secreting mechanism. It might also be suggested that the excretory activity regulated the amount and distribution of the blood flow. On the other hand, the researches of Kahlenberg may lead us to a new physical conception of semipermeable membranes, and of diffusion, and may thus open up for us entirely new conceptions in regard to animal membranes and the role that osmosis plays within the body.

I feel myself unfortunate that in this review of the kidney's functions I have not been able to indicate in any way the probable cause and method of the treatment of uremia.

The futility of trying to form soluble compounds of uric acid, and so aid its being washed out of the kidney, seems to me to be disclosed in the above. Even the highly soluble piperazin compound, or, as shown by Nicolaier lately, the formaldehyde compound, are not excreted as such, but are broken down and excreted as less soluble urates. Probably any beneficial action that piperazin had was due to its action in dilating the kidney vessels and so increasing the blood flow through the kidney.

That digitalin and the purin bodies at times fail is not astonishing when one considers that neither causes a specific secretion, but their action is limited by the state of the bloodstream and its richness or poorness in water.

Further, the inability and failure of digitalis and of caffeine to cause diuresis even in œdema, when salt retention is present, is abundantly evident. It is in those cases that decreased in salt intake has given such astonishing results.

That salines, acetates, and sulphates, etc., act as diuretics when caffeine and calomel fail, is not astonishing when one realizes the changes in blood and in composition of urine which occur when they are exhibited. That they may fail when caffeine is efficient is equally lacking in peculiarity.

Most important of all, I think, is the fact that with our lack of knowledge of the kidney, and how it carries on its function, it is not to be expected that we will be successful, as indeed we are not, in forcing the kidney to excrete when it is diseased. The fact that when there is chloride retention an increased intake may cause more marked retention, taken altogether with the facts mentioned above, that Riedel, Brandenstein and Achard have shown that undue amounts of water cause damage to the kidney, as does excretion of undue amounts of salts, as shown by Leopold, should lead us, especially in nephritis, to guard the kidneys from labor of every kind that can be avoided.

OTITIC BRAIN ABSCESS.[1]

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The study of cases of intracranial suppuration complicating otitis media is always interesting. It has been my pleasure to bring before the society at various times cases of this character. In presenting 2 cases at this meeting, I have taken the liberty of appending an analysis of 102 cases of cerebellar and 100 cases of cerebral abscess, with special reference to the symptomatology, the route of infection, the presence or absence of optic neuritis, and the value of various methods of operative procedure. Unfortunately my own 2 cases terminated fatally. One was a case of cerebellar abscess and the other a case of abscess in the inferior frontal convolution.

Cerebellar Abscess. The case of cerebellar abscess occurred in a young boy, thirteen years of age, who entered the hospital suffering from what seemed to be an acute exacerbation of a chronic suppuration involving the right ear. The patient had severe pain, some œdema over the mastoid, extending down into the neck, a discharge from the ear, and some headache. A radical operation was done upon the right ear, and an epidural abscess over the sinus was found. No grafting was employed. The patient did well for two days, when he was found to have a polymorphonuclear leukocyte count of over 84 per cent. and a leukocytosis of 33,600. There was also some elevation of temperature, and on dressing the wound a suppurating sinus was found extending down the neck. This was drained by a counter-opening and the patient seemed to progress satisfactorily for a day or two, when his temperature again rose to about 104°, and he became lethargic, stupid, and complained of some headache. An examination showed no muscular paralysis, and the optic disks normal. Owing to the fact that there was no evidence of suppuration within the cerebrum, and because there had been an epidural abscess about the sinus at the time of the primary operation, it seemed to me that the most probable site of suppuration was an abscess of the cerebellum. The patient was again placed upon the operating table, and an incision made in the cerebellar dura in front of the lateral sinus. Considerable turbid serum was evacuated, and a probe could be passed into an abscess cavity in the cerebellum. The cerebellum was then explored behind the sinus, the dura incised, and a director inserted into the posterior opening could be made to touch a probe passed into the opening in front of the sinus. Gauze packing was introduced into both openings, and the patient returned to bed. He died within twenty-four hours after the operation.

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[1] Read at a meeting of the American Otological Society, Washington, D. C., May 7, 1907.

In this case there had been absolutely no evidence of intracranial suppuration until the patient's temperature rose suddenly and he became dull and stupid. Without question, the cerebellar abscess had existed for some time. Had symptoms been present at the time of the radical operation undoubtedly he might have been relieved by operation.

As we can only learn the symptomatology of a given disease from the study of the histories of a large number of cases, I thought that it might not be out of place to analyze 102 cases of cerebellar abscess with special reference to the symptomatology. These cases have been collected for me by my assistant, Dr. Charles E. Perkins, to whom I wish to express my sincere thanks.

Route of Infection. In a study of these cases the first point to be noticed is the method of infection. The route of infection was through the petrous portion of the temporal bone in 30 cases, through the lateral sinus in 30, in 4 the infection seemed to travel through the mastoid, in 3 the cerebellar abscess was secondary to a cerebral abscess, while in the remaining cases the route of infection could not be made out. It would seem, therefore, from these statistics, that cerebellar abscess occurs with about equal frequency as the result of infection of the lateral sinus and of infection of the petrous portion of the temporal bone, that is, through the internal auditory meatus, or through the aquæductus cochleæ and aquæductus vestibuli. The cases secondary to an abscess of the cerebrum are, naturally, exceedingly rare.

Symptoms. When we come to the consideration of the general symptoms, it is interesting to note that the most prominent and constant symptom is headache. This occurred in 71 of the cases, and was seldom localized; it was simply complained of as a general headache, and was not referred to any particular region of the skull.

Another prominent symptom of cerebellar abscess is vomiting, which occurred in 54 cases, in which a record is made of the presence or absence of this symptom. In 1 case the notes state that there was "no vomiting." In 1 of the cases which came under my own observation vomiting was present, although it was not very severe, and occurred rather early; in 3 other cases it was not noted.

Vertigo was present in 30 of the 102 cases. In 5 of these the patients had a tendency to fall to the side opposite the lesion. This was present in the case which I reported last year. In the case which I have just reported the patient could not walk at the time when the cerebellar symptoms first appeared, and consequently the character of the vertigo is not noted.

Nystagmus was noted in 17 of the cases. This symptom would therefore, seem to be a rather uncommon complication of cerebellar abscess.

The pulse was slow in 40 of the cases in which any record of the pulse is given. The slow pulse would naturally be an indication of an intracranial complication, and the pulse rate could not be taken as a differential sign in locating the abscess in the cerebellum or in the cerebrum. In 26 of the cerebellar cases the temperature was subnormal, while in 5 the temperature was high.

Retraction of the muscles of the neck was noted in 12 cases.

Stupor occurred in 44 of the cases. This stupor varied from a mental lethargy to a state of hebetude, and, in marked cases, to coma. This symptom is ordinarily present, to some degree, in all cases of intracranial suppuration, the degree of the stupor usually depending upon the degree of intracranial pressure.

The condition of the muscular reflexes was noted in 10 cases only. In 6 the knee-jerk was increased on the affected side, and in 1 case there was slight increase on the opposite side. In 3 cases the knee-jerk was absent. In the other cases the condition of this reflex is not noted.

Regarding the condition of the pupils, in 9 cases the pupils were noted as "unequal." In 5 the pupil on the affected side was dilated, and in but 1 case was the opposite pupil dilated.

Strabismus was noted in 9 cases. In 1 case the eyes were turned to the opposite side, in 7 the character of the strabismus was not noted, and in 1 case it is marked as "internal" strabismus. As the sixth nerve is the nerve usually affected in these cases, strabismus, when present, is usually internal.

Respiratory symptoms were noted in 9 cases. In 3 respiration is noted as "Cheyne-Stokes," while in 6 respiration ceased at the time of operation, and it was necessary to complete the operation under artificial respiration.

Regarding the condition of the fundus oculi, out of the series of 102 cases optic neuritis was present in 34 cases, absent in 37 cases, and in 31 cases the condition of the optic papillæ is not stated.

The analysis of these symptoms, while perhaps not of great value, will, I think, serve to establish more clearly in our minds the characteristics of cerebellar abscess.

Mortality. Regarding the mortality of cerebellar abscess, out of the 102 cases death occurred in 69, while 33 patients recovered.

Operative Treatment. The method of operation practised in these cases was as follows: Out of the 102 cases collated, in 45 the abscess was opened behind the lateral sinus; 25 of these resulted in cure, and 20 in death. In 11 cases the abscess was opened in front of the lateral sinus; in 4 of these cure ensued, and in 7 death occurred. In 46 cases the method of operation was not stated; in 4 of these cases cure ensued, and in 42 death resulted.

The method of operation in any of these cases must depend upon the probable route of infection. I say "probable," because in many cases the avenue of infection can only be surmised. I think we can lay it down as a fairly good rule, however, that, unless at the time of operation the surgeon can trace the route of infection from the lateral sinus, an exploratoy opening in the cerebellum should be made anterior to the sinus, in view of the fact that if evidences of infection by way of the sinus are absent the most probable channel of infection has been either through the internal auditory meatus or through either the aquæductus vestibuli or the aquæductus cochleæ. In either of these latter events the abscess could be more easily evacuated by incising the cerebellar dura in front of the lateral sinus. In some cases the lateral sinus is located sufficiently far backward to permit of a free incision in the cerebellar dura in front of it. In other cases, in which the sinus lies far forward, it is almost impossible to make an incision of any length through the cerebellar dura in front of this vessel, and the posterior route must be chosen. In a number of cases—as in the case reported herewith—it may seem wise, after evacuating the abscess by the anterior route, to make a counteropening through healthy cerebellum behind the sinus, in order to secure perfect drainage.

Cerebral Abscess. I have also to report a case of cerebral abscess which occurred in my practice two years ago. The patient was a woman, aged forty-seven, who gave a history of an acute inflammation of the left ear ten years previously. There had been no aural symptoms during this interval until about two weeks before I saw her, when she had an attack of the grip, accompanied by pain in the left ear. The left ear had been discharging about ten days prior to the time I was called to see her. Upon my examination, the left drum membrane was found to be bulging and red. There was a small perforation in the upper portion of the drum membrane, affording insufficient drainage. There was no evidence of any previous middle-ear suppuration, as far as I could determine from my examination of the ear. At the time of my first examination the left mastoid was excessively tender. A free myringotomy was performed. A smear of the discharge showed the Friedländer bacillus. As the mastoid tenderness did not disappear in spite of incision of the drum membrane, a complete mastoid operation was done forty-eight hours after I first saw the patient. The mastoid cells were found to be extensively involved and the lateral sinus covered with granulation tissue. The sinus wall was extremely thin, and just below where it was covered with granulations the sinus was accidentally wounded in removing the overlying bone. The hemorrhage was easily controlled and the mastoid wound dressed in the ordinary manner. The patient did perfectly well for six weeks, at which time the mastoid wound had almost entirely healed. During this period the temperature had been practically normal, and aside from the fact that she did not gain flesh after the operation, there were absolutely no unusual symptoms marking her convalescence. One month after the operation, following a dressing, the temperature rose to 100°. The following day she suddenly became aphasic and the temperature rose to 102.5°. The patient was seen by several eminent physicians—one a prominent neurologist—and all agreed that the symptoms were due to thrombosis of a branch of the middle cerebral artery supplying Broca's convolution and the island of Reil. Ophthalmoscopic examination was negative. An examination of the blood showed 84 per cent. of polymorphonuclear cells, with a leukocytosis of 11,400. The temperature ranged between normal and 102° for one week, then dropped to 100°, and, although the aphasia did not improve, the general condition of the patient seemed to be better. Owing to the high polymorphonuclear count, I was inclined to believe that the patient was suffering from a cerebral abscess, and a prominent surgeon was called in consultation. He rather shared my opinion, but did not consider the symptoms positive enough to warrant exploratory operation. Blood counts, at frequent intervals, showed a leukocytosis varying from 11,000 to 17,000 and a polymorphonuclear count ranging from 79 per cent. to 85 per cent. After several consultations it was decided not to operate, and, consequently, no operation was performed until the day before death. Two days before her death the patient had a severe chill, complained of headache, and the temperature rose to 101°, but fell the next morning to normal. The day before operation the temperature was normal, but the patient complained of considerable headache. She, however, was perfectly rational. On the evening of this day the temperature began to rise, the patient complained of severe headache, and on the morning before death she had a temperature of 104°. I operated immediately, and found an abscess in the inferior frontal convolution and the island of Reil. Symptoms of meningitis were present when the patient was put upon the table, and I regarded the condition as absolutely hopeless when the operation was performed. An examination of the abscess cavity showed this to be very large, and involving the frontal lobe. The abscess was so large that it could not possibly have developed during the time that the patient was under my care. I am, therefore, inclined to believe that she was suffering from a latent brain abscess, which became re-infected at the time of her acute otitis. Had the operation been performed earlier in this case, I think the chances for recovery might have been good.

The symptomatology of cerebral abscess is far from clear. Through the efforts of my assistant, Dr. Chas. E. Perkins, I have been able to analyze 100 cases of cerebral abscess, with special reference to symptomatology. Some of my own cases are included in this list, but the report embodies chiefly the work of other observers, as most of my own cases have been reported from time to time, and have been intentionally omitted from this list.

Out of these cases, the suppuration was on the right side in 41 cases, on the left side in 42; both ears were involved in 1 case, and in the other cases the side is not reported; 20 followed an acute middle-ear suppuration and 77 followed a chronic middle-ear suppuration. In the remaining 3 cases the duration of the suppuration is not reported.

The *route of infection* in these cases was as follows: Through the tegmen tympani in 40; secondary to epidural abscess in 6; secondary to sinus thrombosis in 6; through the mastoid antrum in 6; secondary to infection through the squama in 2, while in 9 no bone defect was found; 1 case was secondary to a cerebellar abscess (Case 48).

Symptoms. Regarding the symptoms, 77 complained of headache and in 44 vomiting was a prominent symptom. The pulse was slow in 37 cases and rapid in 1. Vertigo was present in 32 cases—a rather unusual symptom for a cerebral abscess. The temperature was high in 7 cases and either normal or subnormal in 20. Coma and stupor were noted in 31 cases. Mental dulness was noted in 20 cases and aphasia in 10 cases. Optic neuritis was present in 32 cases, absent in 20 cases, and in the remainder the condition of the optic disk is not mentioned. Nystagmus was present in 4 cases. Pupillary symptoms were present in 17 cases. General convulsions were present in 5 cases. Paralytic or paretic symptoms were present in 17 cases. In 15 cases the motor disturbance was on the side opposite the brain abscess; in 2 it was on the same side as the abscess. The degree of motor disturbance varied, from slight impairment of motion to hemiplegia. I have seen hemiplegia occur in one or two of my own cases, and it has also been noted in the reports of one or two other cases embodied in this article. In my own case, and in one of the others reported, the hemiplegia only came on after operative interference. Therefore, it might quite as well have been due to a traumatism inflicted at the time of the operation as to the original abscess. In one case, however, namely, that reported by Hansberg,[2] there was complete hemiplegia of the opposite side.

The reflexes were abolished in 1 case and exaggerated in 7.

Operative Treatment. The results of operation show 52 cases resulting in cure and 48 fatal cases.

It is interesting to note the method of operation in these cases. In 41 the abscess was opened through the tegmen, and of these 27 resulted in cure, and 14 in death; 37 cases were opened through the squama, and of these 18 resulted in cure, and 19 in death. In 22 cases the method of operation is not mentioned, and of these 7 resulted in cure, and 15 in death.

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[2] Zeitschrift f. Ohrenheilkunde, 1903, p. 354.

These reports are fairly accurate, although in some cases in which the operator opened first through the tegmen and then, later, extended the operative opening to the squama, the case has been reported sometimes as "squama" and sometimes as cases opened through the "tegmen."

It would seem from this series of cases that the best results are obtained when the abscess can be drained through the tegmen. This undoubtedly depends upon the fact that when the abscess has been drained through the tegmen tympani the operator has been able to open the abscess along the avenue of infection. When this can be done—as has been pointed out by Macewen and Ballance—there is very little danger of secondary meningitis, and very little danger of hernia cerebri, because, as the result of the infectious process, the meninges become soldered together and the subdural space obliterated over a given area. If the incision through the dura and into the brain substance is made through the membranes which have thus become adherent, there is very little chance of secondary meningitis following. If, however, the abscess is opened through the healthy dura, there is great danger of secondary meningitis, which, in the majority of cases, proves fatal. In 3 of my own cases, not included in this series—in which recovery ensued as the result of operation—in 1 case only was the abscess drained primarily through the squama. In 2 other successful cases the combined method was used—that is, the tegmen tympani was removed and the opening enlarged upward and outward into the squama, thus securing more room. In 1 of these cases drainage was made directly along the course of the path of infection, and recovery followed. This patient was seen several years later, and was perfectly well. In a second patient operated upon by one of my assistants during my absence the same plan was followed, and although the dura did not seem to be diseased, the rapidity with which the patient recovered would seem to indicate that the subdural space must have been obliterated as the result of a previous inflammatory process. In 1 patient operated upon by myself, and who made a perfect recovery, drainage was instituted through the squama, and in this instance the membranes appeared perfectly healthy. There was quite a large hernia cerebri in this case, but it was finally overcome, and the patient made a complete recovery.

These statistics would go to show, therefore, that in cases of brain abscess the operator should first search carefully for the path of infection through the tegmen tympani and tegmen antri, and if such a path is found the diseased bone should be removed. If more space is needed the opening may be enlarged upward and outward through the squamous plate of the temporal bone. In incising the dura this incision should be made through the diseased dura, if possible, as here the membranes will have become adherent. In the absence of any evidence of caries or necrosis of the tegmen tympani or tegmen ant i, the bone should first be removed here and the dura exposed, as we may find diseased dura underlying apparently healthy bone. Our statistics also show that, in the majority of cases of temporosphenoidal abscess of otitic origin, the infection takes place in one or the other of these regions. The exploration should, therefore, begin here in every case. Such an exploratory opening, if more room is needed, may be enlarged upward and outward through the squamous plate of the temporal. Unless the symptoms are very urgent, I should be inclined, in future cases, in which the dura appears perfectly healthy, to adopt the suggestion of Mr. Ballance, and, after exposing the dura over a proper area, to incise this and pack the margins of the wound firmly with iodoform gauze, deferring an exploratory incision of the brain substance for from twelve to twenty-four hours. This procedure would serve to relieve the tension, while, at the same time, it would cause an amalgamation of the various layers of the membranes of the brain, thus obliterating the subdural space. Such a soldering together of the membranes would occur in the course of twelve to twenty-four hours. The brain substance could then probably be explored with comparative safety, and the abscess evacuated without much danger of secondary meningitis. When the condition admits of delay, I believe that this plan of procedure will be the proper one to follow.

The various methods of securing drainage in a brain abscess have been discussed so many times that it is quite unnecessary to review them here.

In appending the following bibliography of cerebellar and cerebral abscess, I wish to acknowledge my indebtedness to my assistant, Dr. Charles E. Perkins, who has so carefully collated these cases for me. His painstaking tabulation of symptoms has made the above review of this number of cases possible.

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62. Percy-Jakins, London Lancet, 1904, No. 1, 290.
63. Caboche, Annales des maladies de l'oreille, etc, part i, xxx, 231.
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66. Barrett and Orr, Intercol. Med. Jour., Melbourne, ix, 319.
67, 68 and 69. Manasse Münch. med. Woch., part i, li, 413.
70. Dundas Grant, Jour. Laryn., etc., June, 1901.
71 and 72. Grunert und Schultz, Arch. f. Ohrenheilk., liv, 87.
73. Ferreri, Archiv. Italiano di Otol., etc., April, 1900.
74. Barr, Arch. f. Ohrenheilk., xlix, 297.
75. Ibid., 300.
76 to 79. Preysing, Ibid,, v, 285.
80. Hansen, Ibid., liii, 241.
81. Grunert und Schultze, Ibid., 1902, liv, 71.
82. D'Hoore, Ibid., lviii, 301.
83. Grunert und Zeroni, Ibid., xlix, 165.
84. Voss, Zeit. f. Ohrenheilk., xlii, 255.
85. Luckstorff und Henrici, Ibid., xliv, 156.
86 and 87. Grunert und Dallman, Arch. f. Ohrenheilk., lxii, 143.
88. Tretóp, La presse oto-lar. de Belge, No. III, 217.
89. Delseaux, Ibid., 1904, 274.
90. Robinson, London, Lancet, part ii, 1904, 1566.
91. Blake, American Medicine, ix, 27.
92. Boley, Int. Centralblatt f. Ohrenheilk., iii, 284.
93. W. C. Bull, Jour. Laryn., etc., London, xx, 25.
94. Steinthal, Wurt. Med. Cor., Bl. No. 8, 1899.
95. F. Miyaj, Wojenno Medizinski, St. Petersburg, August, 1904.
96. W. Kummel, Beitrage zur Ohrenh., Berlin, 1905, 313.
97. Roughten, London Lancet, part ii, 1905, 1597.
98. Crampolini, La Clinica Moderne, ii, 344.
99. Dench, Arch. Otol., xxxi, 41.
100. Dench, Trans. Amer. Otol. Soc., part ii, vii, 391.
101. Dench, Ibid., 1906, 266.

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2. Knaggs, London Lancet, part ii, 1903, 806.
3. Wolfers, St. Petersburg med. Woch., No. 1, 1904.
4. Guttman, Arch. Otol., xxxiii, 176.

5. Jordan, Ibid., xxxiii, 186.
6. Kobel, Ibid., xxxiii, 515.
7 to 12. Ucherman, Ibid., xxxiii, 416.
13 and 14. Voss, Ibid., xxxiii, 491.
15. Bacon, Ibid., xxxiii, 269.
16. Wakefield, Ibid., xxxiii, 271.
17. Suckstorff und Henrici, Ibid., xxxiii, 13.
18. Gruening, Ibid., xxxii, 295.
19. Ibid., Trans. Amer. Otol. Soc., viii, 210.
20. Ibid., Jour. Laryn., etc., xiv, 357.
21 and 22. Richardson, Arch. Otol., xxxii, 451.
23. Lewis, Ibid., xxxi, 340.
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25 and 26. Manasse, Ibid., xxx, 328.
27. McKernon, Ibid., xxx, 23, 392.
28. Ibid., Annals of Laryn., xiv, 52.
29. Lewis, Arch. Otol., xxx, 491.
30. Stenger, Ber. klin. Woch., 1901, 292.
31, 32, and 33. Röpke, Arch. Otol., xxx, 30.
34. Heine, Ibid., xxxii, 292; Arch. f. Ohrenheilk., lxi, 151.
35, 36, and 37. Ibid., xlv, 269.
38 to 41. Voss, Deut. med. Woch., 1903, 290.
42. Walker Secker, Jour. Laryn., etc., xxi, 157.
43. Hammond, Boston Med. and Surg. Jour., cliv, 102.
44, 45, and 46. Whitehead, Jour. Laryn., etc., xx, 84; xxi, 348.
47. Tretôp, Ibid., xxi, 391.
48. Neuman, Ibid., xxi, 259.
49. Halstead, Ibid., xx, 611.
50, 51, and 52. De Greift, Ibid., xix, 489.
53. Delseaux, Ibid., xix, 485.
54. Randall and Potts, Arch. Otol., xxxii, 177.
55. Turner, Jour. Laryn., etc., xix, 563.
56. Brady, Ibid., xix, 339.
57. Dundas Grant, Ibid., xvii, 145.
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60. De Santi, Ibid., xvii, 268.
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63. Angustowski, Arch. f. Ohrenheilk., lvi, 145.
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78 and 79. Grunert und Dallman, Ibid., lxii, 96, 146.
80 and 81. Hansberg, Zeit. f. Ohrenheilk., xliv, 352.
82 to 85. Heiman, Klin. Vorträge aus dem Gebiete der Otologie und Pharyngo-rhinologie, Haug, v, 155, 159, 160, 170.
86. Hölscher, Arch. f. Ohrenheilk., lii, 165.
87. Jaboulay and Riviere, Ibid., xliv, 298.
88. H. Knapp, Ibid., lvi, 138.
89. Lehr, Ibid., li, 71.
90. Lermoyez, Ibid., lix, 151.
91, 92, and 93. Meier, Ibid., xlv, 152.
94 to 96. Preysing, Ibid., li, 75.
97. Dench, Trans. Amer. Otol. Soc., vii, 57.
98. Ibid., N. Y. Eye and Ear Infirmary Reports, January, 1897, 120.
99. Ibid., Arch. of Otology, xxvii, 247.
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ACTINOMYCOSIS OF THE SKIN OF THE FOOT.

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Primary invasion of the skin and subcutaneous tissues by Actinomyces bovis is of such rare occurrence that I deem it not unwise to add another instance of this affection to the very few that have already been recorded in the literature. If we glance at the statistics of those who have made a special study of this subject we find that the cases number less than 20, and a careful perusal of the histories makes it evident that even some of these may have been due to secondary rather than primary invasion of the skin. Illich[1] was able to find but 11 cases in his series of 421. In the 58 American cases collected by Ruhräh[2] there were but 3 of primary skin affection. In a total of 628 cases from all sources the same author reports a total of 16 of the skin. Von Baracz[3] does not mention its occurrence in his large personal collection of 60 cases.

Leser[4] was the first to arouse interest in this particular localization by the publication of 3 cases. From a careful clinical and microscopic study he was able to distinguish two general types, an ulcerating and a nodular form. The first he described as being characterized by the formation of granulation tissue, by necrosis, and by the production of ulcers. The second commences with the appearance of a nodular or papular eruption which has a tendency to peripheral extension and central cicatrization similar to that occurring in lupus vulgaris.

According to most of the observers the affected skin is bluish or has a violaceous tint. There is a formation of nodular protruberances which coalesce, break down in places, and finally perforate so as to form chronic sinuses or indolent ulcers. Leser calls particular attention to the absence of involvement of the regional lymphatic nodes, and considers this of diagnostic importance.

If we review the histories of a number of the reported cases we note, first, that many of them represent extensive lesions involving not only the skin, but the fascia, muscles, and even the bones, and second, that certain cases of cutaneous actinomycosis of the face may be secondary to buccal invasion of the organism. If we subtract these from the total number, but very few instances of true primary cutaneous involvement remain. For the sake of reference, I shall briefly summarize the important data belonging to the histories of most of the reported cases.

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[1] Klinik der Aktinomykose, Wien., 1892

[2] Annals of Surg., 1899, xxx, p. 417.

[3] Ibid., 1903, xxxvii, p. 336.

[4] Archiv f. klin. Chir., xxxix, p. 722.

Case I.—Bertha:[5] Male, aged sixty-six years; thrasher. A small nodule appeared on the dorsum of the right hand; gradual enlargement and conversion into an ulcer. After eight months the ulceration had extended over one-third of the back of the hand. The surface of the ulcer is nodular, in places covered with a thin, livid epidermis; there are a few sinuses. The process is limited to the skin and subcutaneous tissues. Treatment: excision of the ulcer. Cure.

Case II.—Bertha: Male, aged forty-five years; laborer on farm. Sustained a wound on the ball of the thumb. Four weeks later there was considerable swelling in the neighborhood of the wound with reddening of the hand. In the course of the next month a number of pea-to-hazel-nut sized foci of pus developed. Treatment: excision of diseased skin; curetting of wound. Cure.

Case III.—Dreyfus:[6] Male, aged ten years; worked on a farm. He has had a swelling of the left mammary region for about six months. Now spontaneous perforation has occurred and ulcers have formed. There is an area about the size of a palm, of infiltrated skin over the breast; there are three ulcers with grayish yellow, pale, sluggish granulations. Treatment: excision; improvement. The author calls it an ulcerating and fungoid type of lesion.

Case IV.—Gross:[7] Female, aged twenty-seven years, very anemic and thin with a suppurative process at the angle of the jaw on the left side. There is a red indurated flat lesion resembling lupus of the skin. The diseased area was extirpated and the actinomyces demonstrated.

Case V.—Köhler:[8] Male, aged fourteen years; peasant. There developed a painful swelling of the whole of the left leg. This became localized to the upper part of the tibia, broke down and perforated after a lapse of one and one-half months. In the neighborhood of the sinus nodular infiltrations of the skin made their appearance and rapidly become necrotic. Upon admission, some three months after the onset of the disease, there was a mass over the upper third of the tibia about the size of a hen's egg. At its lower pole there was an ulcer at the bottom of which the exposed bone could be seen. Treatment: excision of diseased portions, removal of exposed bone. The inner tuberosity of the tibia was filled with a soft tissue in which numerous colonies of actinomyces were found. Improved, but not wholly cured two years after operation.

Case VI.—Leser: Male, aged thirty-five years; merchant. Disease began with the formation of an inflammatory swelling a little below the middle of the back of the left forearm; ulceration of the skin then occurred; edges partly undermined, irregular, hard, surrounded by a ring of induration. Similar lesions were found in the following places upon admission to the clinic: (1) An ulcer on the right side of the head, almost the size of a silver dollar; (2) a lesion closely resembling that of lupus in the right infraclavicular fossa where there was an indurated area of skin presenting about a dozen lentil-sized, red-brown nodules; and (3) on the left side of the abdomen an ulcer similar to the one on the arm. Treatment: curettement, cauterization, excision. Cure.

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[5] Wien. med. Woch., 1888, xxxv.

[6] Münch. med. Woch., 1903, l, p. 2291.

[7] Soc. de méd. de Nancy, 1896 (cited by Poncet).

[8] Charité Annalen. 1887, xii.

CASE VII.—Leser: Male, aged thirty-four years; merchant. For the past six days he has had a swelling of the skin of the right calf. Three days ago chills and headache. Fluctuation easily detected, no involvement of lymph nodes. Incision, drainage, later removal of portion of underlying muscle. Cure after four months.

CASE VIII.—Leser: Male, aged twenty-nine years; locksmith. In the neighborhood of a recently-healed burn on the upper part of the right leg there developed a painful swelling of the skin with several purulent areas. Ulceration soon occurred. Two such areas were separated by indurated, undermined skin. Upon curetting the ulcers an indurated process was found to extend deep down into the fascia. Treatment: excision of diseased areas; cautery. Cure.

CASE IX.—Lührs:[9] Male, aged thirty-seven years. A hard tumor on the outer side of the popliteal region. Incision and evacuation of serous fluid, granulation tissue, and "sulphur" granules. Treatment: incision; drainage. Cure.

CASE X.—Lührs: Male, aged fifty years; laborer on farm. Behind the left sternocleidomastoid there is a hard, painless nodule which upon incision showed the typical actinomycotic granules. Treatment: incision. Cure.

CASE XI.—Milian:[10] The history of this case is that given by Hudelo, Gaston, and Lebar, when they presented the patient to the French Society of Dermatology. Their diagnosis was tuberculosis. Milian, however, had seen the case almost two years before and by microscopic examination had established the diagnosis of actinomycosis.

Male, aged fifty years; porter. Onset of disease, twelve years ago. On the sole of the foot near the inner border there was a slightly painful nodule about the size of a lentil. This remained stationary for several years. Five years ago little nodules appeared along the inner border of the foot. These became softened and discharged thin pus. When seen by the authors the following condition was noted: The skin over the inner surface of the foot was bluish, beset with nodules of a violaceous tint, some hard, others soft and closely resembling angiomas. Some of these had evidently broken down. Microscopic examination threw no light upon the nature of the process.

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[9] Inaug. Diss., Gottingen, 1889.

[10] Bull. soc. franc. de dermat., 1905, xv.

Case XII.—Monestie:[11] Female, aged thirty-six years. When seen by the author the patient had a tumor of the right submaxillary region. Seven months previously the disease began with a little nodule in the skin which gradually increased in size. After a period of four weeks the tumor ulcerated and evacuated some blood. Ever since there were intermittent discharges of pus and blood. At the time of examination she had an elongated, dark-red, or violaceous tumefied area in the skin, which showed areas of softening. About this there was a zone of induration. The teeth and jaws were healthy.

Case XIII.—Müller:[12] Female, aged twenty-eight years. Two years previously patient had run a wooden splinter into the palm of her right hand near the base of the middle finger. The splinter healed in leaving a painless nodule. This became suddenly reddened without apparent reason. Incision revealed a cavity filled with granulations and a small splinter which was overgrown with actinomyces colonies. Cure.

Case XIV.—Nocard and Lucet:[13] Male. Following the kick of a horse an abscess developed in the upper part of the left thigh. Perforating sinuses remained.

Case XV.—Partsch:[14] Male, aged sixty years. Two months after the removal of a carcinoma of the breast two sinuses formed together with purulent areas in the scar. A small hazelnut-sized cavity filled with granulation tissue was found in the old scar. In this the actinomyces were demonstrated. The infection probably occurred at the operation. Treatment: curettage; excision of scar tissue. Cure.

Case XVI.—Poncet:[15] Female, aged thirty-two years, was seen for an affection of the left cheek which had lasted four months. She first noticed a little nodule just below the angle of the mouth. The skin over it became red and then ulcerated. In spite of the extraction of a premolar which was supposed to have caused the lesion, a number of similar nodules appeared in the vicinity. At the end of three months the deeper parts became infiltrated, there was marked trismus and œdema extending into the parotid region. The cutaneous lesions at the time of admission to the hospital about four months after the beginning of the disease, are thus described: "toute la région comprise entre la commissure labiale et l'oreille, est recouverte d'élevures hémisphériques confluentes de 1 á 3 centimètres de diamètre, de 1 centimètre de hauteur. Leur sommet présente des cratères qui laissent échapper du pus contenant des grains jaunes actinomycosiques. La peau, á leur niveau, a une coloration rouge muqueux."

Case XVII.—Tusini:[16] Female, aged thirty-four years. At the age of ten she sustained a slight wound of the left leg with a pitch-fork. The wound healed and left a small scar. Ten years ago a few nodules appeared in the old scar. These grew gradually and discharged pus, leaving ulcers which slowly healed. Fresh nodules were repeatedly formed in the vicinity, spreading gradually over a large area until finally the whole of the left leg was involved. At the time of examination of the patient, Tusini found 43 ulcers and numerous sinuses. Some of these could be traced down as far as bone. The muscles of the leg were almost completely converted into fibrous tissue.

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[11] Thèse Paris, 1895, (cited by Poncet).

[12] Beitr. z. klin. Chir., 1888, iii, p. 355.

[13] Acad. de méd., 1888 (cited by Poncet).

[14] Breslauer aerzt. Zeit., 1881.

[15] Deut. Zeit. f. Chir., 1886, xxiii, p. 497.

[16] Arch. f. klin. Chir., lxii.

These 17 cases have been either reported or cited as being instances of primary actinomycosis of the skin. However, it will be seen at once that those recorded by Poncet, Monestié, and Gross, although they first showed lesions in the skin, could, according to their situation, have been secondary to infection by way of the mouth. It is indeed difficult to determine whether we are dealing with a primary skin lesion in the cases that occur in the cervicofacial region. And thus the old term "Actinomycosis cutis faciei" has justly fallen into disuse because of this very fact. The cases published by Köhler, Nocard, Lucet, Müller, and the first one of Lührs were evidently more deeply-seated affections, but caused by infection through the skin. If we are to include such examples as that cited by Müller (foreign body under the skin) under the actinomycotic lesions of the skin we would have to add the more recently reported instances of panaritium due to the same cause.[17] This leaves then but a very few cases, those of Bertha, Dreyfus, Milian, Partsch, Leser, and Lührs in which a primary actinomycosis of the skin was the main lesion.

The striking point noted by many of the observers is the resemblance of the skin lesion to some form of tuberculosis. Leser was the first to call attention to this and showed the importance of a thorough microscopic study. In the case in which Milian made the diagnosis we have another example of the difficulty in recognizing the disease. Hudelo[18] as well as most of the members of the French Society for Dermatology at a meeting of which the patient was shown, looked upon the case as most probably tuberculous.

In my own patient the presumptive diagnosis of an inoculation tuberculosis was made. The patient was a young girl who came to the Good Samaritan Dispensary complaining of a "wound" on her foot behind the toes.

S. B., Russian; female, aged sixteen years; operator, consulted me at the dispensary on March 4, 1907. Four months previously she noticed a tiny "pimple" on the back of her foot just behind the fourth toe. She does not remember having injured or wounded her skin in any way. She has lived in this country for eight years and always in New York City. The small red spot gradually enlarged until it attained its present size. It causes her little pain, but considerable discomfort owing to the pressure of her shoetops. She has always been perfectly well.

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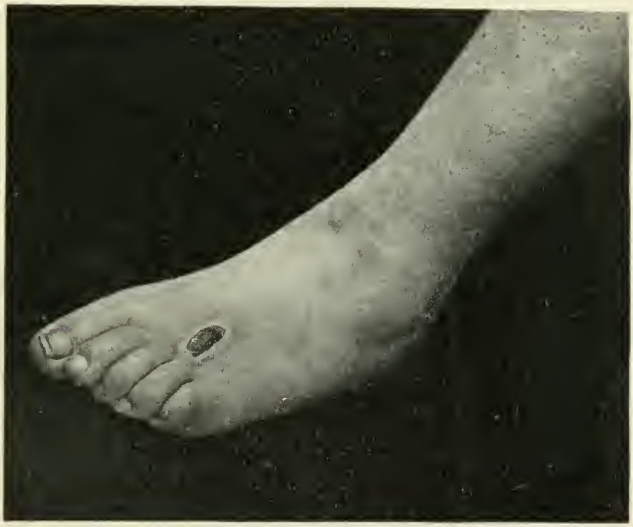
[17] Thevenot, Massaglio and Dor.

[18] Bull. soc. franc. de dermat., 1901, p. 358.

The patient is a well-nourished, somewhat undersized but well-developed girl. Lungs, heart, and abdomen are negative. On the dorsum of the left foot just behind the interval between the third and fourth toes there is an irregularly elliptical tumefied area in the skin, 2.5 x 1.3 cm. (see figure). The skin is elevated about 4 mm. reddish-blue or violaceous in color, the epidermis intact but apparently thinned, particularly in several places where there seems to be a tendency to perforation. The whole area is soft to the touch and semifluctuating, its margins rise fairly abruptly and are surrounded by a slightly indurated zone. The general appearance is not very unlike an infected angioma. The femoral and inguinal glands are not enlarged.



Actinomycosis of the skin of the foot.

The area was circumscribed under cocain anesthesia, sliced off, and the base thoroughly curetted. The latter was found to be hard, apparently made up of dense connective tissue. Upon removal, a few drops of bloody pus were seen to come from the deep parts of the tissue. A number of fresh cuts were made through the lesion but nothing suggestive of sulphur granules was detected. The small number of actinomyces colonies (three in all of the tissue examined) explains the fact that they were overlooked.

Practically all of the diseased tissue was divided into pieces and examined. Twelve blocks were made and in only one of these were the actinomyces found. The general picture is that of a productive inflammatory process with areas of purulent infiltration in the neighborhood of the parasites. Save for the epidermis which is intact over the greater part of the affected area, very little of normal corium or stratum subcutaneum is left. These are replaced by young connective tissue in various stages of development. The epidermis is thinned in places; here and there the stratum mucosum is invaded with leukocytes, shows necrosis and degeneration of the cells. The actinomyces colonies are but three in number. Two of these lie about 1 mm. below the surface, the third 1 mm. deeper. The threads stain well with the Gram method and the clubbed ends are well developed, staining intensely with acid fuchsin. Around each colony there is a zone of polynuclear leukocytes. The deeply-situated fungus is imbedded in rather old connective tissue.

A very striking feature is the large number of newly-formed blood-vessels. The connective-tissue cells or fibroblasts, are frequently arranged concentrically about these vessels for varying extents. Often these are succeeded by collections of plasma cells. These are very numerous and are to be seen not only in the vicinity of the vessels but scattered throughout the tisue. In the superficial layers there are many cells containing brown pigment granules. A few of these are to be seen scattered irregularly throughout. In general the connective tissue is very rich in cells. The sections made from the periphery of the lesion show connective tissue of an older type. Here the plasma cells and fibroblasts are few in number, the intercellular substance is in abundance and many elastic fibers are found.

There has been hemorrhage and necrosis in a number of areas. In one place there is a collection of giant cells grouped in a circular manner. Enclosed by them there are a few leukocytes and some hyaline spherical bodies which stain poorly with all the dyes. Whether these are remnants of some foreign body I was unable to prove with certainty. The presence of the giant cells, however, is suggestive.

During the first seven or eight days that the patient was under observation the granulations that had formed looked sluggish and unhealthy. The patient was given potassium iodide after the sixth day. In the second week there was marked improvement and after twenty-four days the wound was completely healed.

We were evidently dealing here with a case of primary actinomycosis of the skin, in which the history gave no clue as to source of the infection. Undoubtedly inoculation must have occurred at some point in the affected area. A careful search for a foreign body proved futile. Perhaps we may group this case under the type described as nodular, although no discrete nodules could be made out. However, the lesion may have been formed by coalescence of a number of smaller ones. The failure to ulcerate is an interesting feature when we consider that four months had elapsed since it was first observed, and the fact that this portion of the foot must have been continually exposed to the pressure of the shoe. Finally, we may call attention to the small number of colonies. A superficial examination of but a few sections would have failed to reveal the true nature of the disease. Leser had made a similar observation in studying his own cases. In Milian's patient this same difficulty led to the probable diagnosis of tuberculosis. Perhaps a more thorough microscopic study of localized lesions of this type may cause us to change our present views as to the infrequency of actinomycosis of the skin.

HEMORRHAGES IN THE COURSE OF BRIGHT'S DISEASE, WITH ESPECIAL REFERENCE TO THE OCCURRENCE OF A HEMORRHAGIC DIATHESIS OF NEPHRITIC ORIGIN.[1]

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The occurrence of hemorrhage as a complication of Bright's disease has been familiar to clinicians since the days of Richard Bright (1827). But even before Bright, Latour,[2] in 1815, called attention to the coincidence of *epistaxis* and other forms of bleeding with dropsy, while the all-observing Morgagni, at a still earlier date, reported the case of a woman in whom, though she already had the odor of urine on her breath, vomiting of blood and bleeding from the nose proved beneficial. Rayer[3] was among the first to appreciate fully the significance of epistaxis as a symptom of Bright's disease. Naumann[4] cites the case of a young man who died in convulsions after having had repeated attacks of violent nose bleed, in one of which he lost as much as six pounds of blood. The autopsy showed enlarged and inflamed kidneys. While admitting that the hemorrhage in such cases might be purely accidental, Rayer was, nevertheless, of the opinion that it was more probably the result of an alteration of the blood, consequent upon the derangement of the urinary secretion.

The close connection between Bright's disease and *cerebral hemorrhage* was strikingly shown in the classical paper of W. Senhouse Kirk,[5] while ophthalmologists have made us familiar with the frequency of hemorrhage into the eye in the course of nephritis.

These various forms of hemorrhage have been attributed, particularly by Huchard,[6] to vascular degeneration and hypertension;

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[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 7, 8 and 9, 1907.

[2] Quoted by Launois, Thèse de Paris.

[3] Traité des maladies des reins, i, 605.

[4] Quoted by Rayer.

[5] Medical Times and Gazette, xi, 515.

[6] Bull. et mem. Soc. méd. de hôp. de Paris, 1888, 3 s., v, 287–290.

but the interesting studies of Schoenemann[7] seem to demonstrate that, as far as nose bleed is concerned, these are not necessary factors. He found after death numerous superficial hemorrhages into the mucous membrane of the nose. The number of peripheral capillaries, that is, those near the epithelial surface, was increased; in many cases these capillaries had broken through the basal membrane, the capillary loops having forced their way through the basal epithelial cells. During life the capillary walls, as well as the epithelial covering, rupture, and the blood escapes upon the surface. Schoenemann did not observe these conditions in diseases other than nephritis. They cannot be regarded as the result of ordinary circulatory obstruction; the capillary walls themselves seemed normal in appearance, and the deeper vessels showed no degenerative changes, such as have been described in other parts of the body. Perhaps the changes are due to a circulating substance which penetrates by osmosis into the surrounding structures and weakens them and the capillary walls in such a way that they cannot resist the blood pressure.

That *metrorrhagia* may be a symptom of nephritis is not so well known, although several writers have called attention to it. Among the older authorities who mention it are Grainger Stewart,[8] and Lecorché.[9] Braun[10] reports 2 cases of metrorrhagia. Delbet[11] has recorded the case of a girl who died as the result of a profuse vaginal hemorrhage, in his opinion, dependent upon renal disease. Bonifas,[12] in an intractable case of uterine hemorrhage in a woman, aged fifty-five years, saw an arrest of the hemorrhage under antinephritic treatment. Quite recently, Violet[13] reported 3 cases of profuse metrorrhagia, apparently caused by interstitial nephritis. He points out, as does Cumston,[14] the importance of recognizing the connection between metrorrhagia and nephritis, as otherwise improper treatment may be instituted. Ergot, in the opinion of both writers, is contra-indicated in such cases.

Hemorrhage from the lungs, as a manifestation of renal disease, is rare. Cases are recorded by Hawkins,[15] Fisher,[16] and Duclos,[17] and the subject is mentioned by Dickinson, Fuerbringer,[18] and Senator.[19] In Hawkins' case, the hemoptysis continued for a year and two months, the autopsy showing cirrhosis of the kidneys. According-ing to Duclos the hemoptysis, which is most frequent in old persons, is more often a precursor than an accompaniment of interstitial nephritis, and is due to sclerosis of the pulmonary vessels. The same cause that produces these changes in the vessels of the lungs also acts upon those of the kidneys, and thus brings about interstitial nephritis.

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[7] Arch. f. Laryng. und Rhin., 1901-2, xii, 437.

[8] Bright's Diseases, New York, 1871, 238.

[9] Traité de maladies des reins, 1875, 398.

[10] Quoted by Wieger (Launois, loc. cit.).

[11] Bull. soc. anat. de Paris, 1885, lx, 183-186.

[12] La normandie médicale, 1898, xiv, 102.

[13] Lyon médical, 1904, ciii, 428-438

[14] Buffalo Medical Journal, 1904-1905, 718.

[15] Trans. Clin. Soc., London, 1891-2, xxv, 237-242.

[16] Bristol Med.-Chir., Jour., 1904, xxii, 234-247.

[17] Rev. gén. de clin. et de thérap., 1890, iv, 329; 345.

[18] Die Krankheiten der Harn- und Geschlechts-Organe, Braunschweig, 1904, 132.

[19] Nothnagel's Encyclopedia of Practical Medicine, American edition, Diseases of the Kidneys, etc., 227.

Fisher, in addition to 1 case of hemoptysis, reports 4 cases of *hematemesis* associated with small, white kidneys. In explanation of these hemorrhages, he propounds an ingenious hypothesis, to which I shall refer later. Another case of severe gastric hemorrhage, in a nephritic patient of thirty, is reported by Bonifas.[20]

Hematuria, a common symptom of acute nephritis, or of the acute exacerbation of chronic nephritis, may arise in the absence of acute manifestations, as in the cases reported by Askanazy[21] and Kusumoto,[22] in which the hemorrhage occurred apparently from the engorged veins of the renal pelvis.

An interesting form of nephritic hemorrhage is that into the *ear,* of which Schwartze[23] has reported a case. Haug,[24] in an exhaustive paper upon this subject, collects a number of cases from medical literature, all of them examples of hemorrhage into the structures of the ear in Bright's disease. Alibert, in 1880, devoted a thesis to the subject of otorrhagia in nephritis. Hemorrhage into the tympanic cavity may occur even before other symptoms of nephritis are manifest. It thus has the value of an early or prenephritic symptom.

I should not fail to mention the fact that Dieulafoy[25] believes that auditory disturbances, such as tinnitus, partial or complete deafness, and otitis media are more frequent in the course of Bright's disease than disturbances of the eye. Pissot[26] found among 66 cases of chronic nephritis, 34 suffering from auditory disorders.

In addition to the isolated hemorrhages I have so far described, we meet in Bright's disease, with a true *hemorrhagic diathesis,* 2 cases of which have come under my own observation. In a sense, the term hemorrhagic diathesis is also applicable to those cases of epistaxis, metrorrhagia, etc., in which the hemorrhage is uncontrollable, and in which there is no local lesion to explain the bleeding.

Medical literature contains but few references to the hemorrhagic type of bleeding in its relation to Bright's disease. Avrad[27] saw a well-marked example in a case of scarlatinal nephritis. An incomplete report of a case was made by Imbert-Gourbeyer,[28] in 1856. Levi[29] reports 2 cases—1 of parenchymatous nephritis with epistaxis and hemoptysis, terminating in recovery; and 1 of contracted kidney, with dropsy, hemoptysis, and epistaxis, ending in death.

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[20] Loc. cit.

[21] Zeit. f. klin. Med., Band lviii, 432.

[22] Deut. Archiv f. klin. Med., 1907, Band lxxxix, Heft 5 und 6, 405.

[23] Arch. f. Ohrenheilk, iv, 13.

[24] Deutsche med. Wochenschrift, 1896, 723.

[25] La France méd., 1877.

[26] Thèse de Paris, 1878.

[27] Gazette médicale, 1853.

[28] Quoted by Launois.

[29] Quoted by Launois.

Among systematic writers, Senator,[30] Fuerbringer,[31] Castaigne,[32] and Osler[33] mention hemorrhagic diathesis as a rare complication of nephritis. According to Wagner,[34] and to Senator,[35] the hemorrhagic diathesis sometimes precedes and is the cause of nephritis. More probably, however, the cause of the one is the cause of the other. A case of this nature is the one reported by George Johnson.[36] A man, aged thirty-seven years, was seized with epileptiform attacks, rigors, and pains in the limbs, followed by a purpuric eruption and general dropsy; the urine was albuminous, and contained epithelial casts and blood. Johnson believed that in this case, which terminated in recovery, the cutaneous eruption and the desquamative disease in the kidneys were primarily consequences of the poisoned state of the blood, and that the dropsy was a secondary result of the renal disease. A case analogous to Johnson's is reported by Girode.[37] Purpura hemorrhagica came on in the course of a chronic diffuse nephritis in an eight-year-old boy. The hemorrhages occurred very early in the disease, and suggested a common, infectious origin for both the purpura and the nephritis.

Samuel Gee[38] briefly mentions 3 interesting cases of purpura in children in association with nephritis. The onset was marked by diarrhœa, with blood in the stools; in 2 cases there was also vomiting. The purpura occurred in repeated accesses, but finally ceased, although the albuminuria persisted. These cases remind one of the erythema group so exhaustively described by Osler; in all probability they belong to it, and it is a fair assumption that the purpura, the diarrhœa, and the renal disturbances were all dependent upon one and the same infectious or toxic agent.

The history of the 2 cases that have come under my observation is as follows:

CASE I.—J. S., male, aged thirty years, a Russian tailor, was admitted to the Polyclinic Hospital on March 21, 1904, in the service of my colleague, Dr. Schneidemann, with a severe inflammation of the eye. The patient, whose faimily history was negative, had had no other illness, aside from measles in infancy, except two previous attacks of nephritis: the first seven years before, characterized by swelling of the feet, and, the second, two years later, in which the face was chiefly swollen. The third had begun three weeks before, with swelling of the face; but he was able to work until a few days prior to his entrance into the hospital. When admitted he was found to be pale and poorly nourished; the face was swollen, more particularly around the chin, the left eye, and the left malar and temporal regions. The left)eye was inflamed and reddened, and the conjunctiva chemotic. The right eye was painful on palpation, and its tension seemed increased. The heart was enlarged, the apex beat being in the sixth interspace in the nipple line; the first sound was muffled; the second much accentuated, with a tendency to reduplication; the radials and temporals were thickened. A blood examination showed: red blood corpuscles, 2,350,000; white blood corpuscles, 15,200; hemoglobin, 50 per cent., with a differential count as follows: polymorphonuclears, 78; small mononuclears, 13; large mononuclears, 4.5; eosinophiles, 3; transitionals, 1.5. There was no ascara; no ascites; no œdema of the legs; no enlargement of the spleen.

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[30] Loc. cit.

[31] Loc. cit.

[32] Manuel des maladies des reins et des capsules surrenales, 1906, 268.

[33] Practice of Medicine, sixth edition, 669 and 713.

[34] Quoted by Senator.

[35] Loc. cit.

[36] Diseases of the Kidneys, London, 1852, 152.

[37] Arch. de phys. norm. et path., 1887, 3 s., ix, 178-185.

[38] St. Bartholomew's Hospital Reports, 1880, xvi, 47.

Dr. Schneidemann made a careful examination of the eye and reported as follows: "When the man came in he had redness and great œdema of both eyelids. Conjunctiva highly congested and chemotic; not tense, but relaxed and folded on itself. The lower conjunctiva was everted, and the upper slightly from the swelling; the motility of the eye was somewhat impaired, especially outward. The right pupil was moderately dilated, larger than the left, and immovable. Cornea was insensitive. Anterior chamber was extremely shallow. Iris nearly in contact with the cornea; tension + 2. A dull fundus reflex; but no details could be obtained. The œdema, mentioned above, extended to the region over the malar bone. On account of the man's condition, the functional testing of the eye for vision was unsatisfactory. There seemed to be some, but rather uncertain, perception for large objects. The patient complained of headache around the orbital, temporal, and malar regions."

On March 24 the patient began to bleed from the mouth, and, on examination, a carious tooth was found, which was thought to have caused a wound in the cheek; but, although the ragged edges of the tooth were broken off and styptic applications were made to the cheek, the bleeding continued. On account of the eye trouble, a leech was applied to the right temple; when it dropped off, there was free bleeding from the wound, a collodion dressing being required to check it.

On the 26th the patient had a convulsion, following which he was bled to the amount of fifteen ounces. After the venesection, he received an intravenous injection of 1200 c.c. of salt solution. Despite this, he had another convulsion on the same day.

On the 29th, a hemorrhagic suffusion appeared on the right side from the third rib to the costal margin, and a few purpuric spots were seen on the right arm. No history of an injury could be obtained, except the hypodermoclysis, which he had had three days before

On the same day he began to have hiccough, and gradually passed into a stuporous condition.

The ecchymosis on the right side became more extensive, and the spots on the right arm increased in number. Blood was running constantly from the mouth, and the breath was very fetid. The urine contained a large amount of albumin (1.5 parts in 1000), red blood corpuscles, and granular and hyaline casts.

On the 31st, a systolic murmur appeared, its area of maximum loudness in the third left interspace. Despite active treatment in the way of hot packs and stimulants, the pulse gradually grew weaker, and on April 1, eleven days after admission, the patient died. No autopsy could be obtained.

Case II.—W. D., tavernkeeper, aged fifty-five years, born in Philadelphia, was admitted to the Jewish Hospital, September 2, 1904. He had never been ill, and denied drinking to excess. He attributed his trouble, namely, hemorrhages from the mouth and bowels, which had begun eight weeks before, to having neglected his own health in attending to his wife while she was suffering from an attack of pneumonia. When admitted to the hospital he was stuporous and delirious, and in evident uremia. The breath was urinous, and, according to the history obtained, he had voided no urine for forty-eight hours. The mucous membranes were pale; the tongue and teeth covered with recent blood clots; the heart sounds clear; pulse slow and feeble; pupils widely dilated. There was no ascites; no enlargement of the abdominal veins. The urine, which had to be drawn, had a specific gravity of from 1010 to 1012; contained albumin, and red and white corpuscles. Blood examination showed: red corpuscles, 4,440,000; white corpuscles, 6680; hemoglobin, 55 per cent.; differential count: polymorphonuclears, 83 per cent.; lymphocytes, 9.6 per cent.; mononuclears, 4.7 per cent.; eosinophiles, 0.6 per cent.; transitionals, 2 per cent.

The man died in a convulsion on September 10, 1904; the bleeding having continued to the end. There were no purpuric spots upon the surface. The autopsy revealed marked anthracosis of the lungs; a moderately enlarged heart without valvular lesion, and a smooth liver of normal size. The kidneys were enlarged and covered with small cysts; the capsule stripped with difficulty. On section, the kidney showed an almost total absence of cortical substance and marked fibrosis; the cut surface bled freely. The pathologist thought that the condition was one of acute exacerbation of a chronic, diffuse nephritis. The gastrointestinal tract and urinary bladder were normal.

There are four ways in which one may interpret the relation of the hemorrhagic diathesis to renal disease:

1. The hemorrhagic diathesis may be an accident; that is, an independent affection not causally related to the nephritis. This would obtain in such cases in which a septic infection, as, for in-

stance, ulcerative endocarditis, coexisted with the renal disease and caused the hemorrhagic state.

2. The hemorrhagic diathesis may be the cause of the nephritis. Cases of this type, as has been previously stated, have been observed by Wagner and Senator. It might be difficult, however, to prove in a given instance that the third explanation does not better apply.

3. The two may be due to the same cause. This relation of the two conditions is fairly clear in a number of cases in the literature, notably those of Johnson, Gee, and Girode, already cited.

4. The renal disease may be the cause of the hemorrhagic diathesis. This is the explanation I would give for my own cases, and is the group to which I especially desire to call attention.

The question arises, in what particular way does the nephritis cause the hemorrhagic diathesis? Several possible etiological factors suggest themselves: (1) Hypertension; (2) arterial disease; and (3) toxemia.

The first and second are important factors in the ordinary types of nephritic hemorrhage. In all probability they are a sine qua non; but they are not of themselves sufficient to cause a true hemorrhagic diathesis, for the production of which another element, presumably a toxin, is necessary. Flexner's[39] studies on the effects of snake venom have shown the existence of substances, by him termed hemorrhagins, that are capable of causing hemorrhagic extravasations through a destructive action upon the capillary endothelium. Toxins with analogous properties may, as Fisher[40] has suggested, be present in the blood in nephritis. The hypothetic poison, whatever it may be, in addition to acting upon the vessel walls, may also impair the integrity of the blood—an impairment similar to that found in chronic jaundice.

Quite recently, Friedeman[41] found in the pancreatic juice a powerful hemolysin possessing the property of dissolving homologous red corpuscles and causing hemorrhages. In health it is neutralized by a protective substance; but we may conceive conditions—among them Bright's disease—in which this substance fails to act. In its power to cause hemorrhage and paralysis, the poison of the pancreas resembles crotalus venom.

Prognostically, nephritic hemorrhages of every variety constitute a sinister omen; the hemorrhagic diathesis in particular seems to presage an early death. It is evident that in every obscure case of hemorrhagic diathesis, as well as in cases of apparently causeless bleeding from mucous surfaces, the possibility of a renal origin should be borne in mind.

CONCLUSIONS. 1. Bright's disease may cause hemorrhage from the nose, uterus, lungs, and stomach; hemorrhage into the brain, eye, and ear; and also a more or less well-marked hemorrhagic diathesis.

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[39] AMER. JOUR. MED. SCI., 1903, 202.

[40] Loc cit.

[41] Deutsche med. Wochenschr., 1907, Nr. 15.

2. The last-named condition is characterized by bleeding into the skin and from the mucous membranes.

3. The true cause of the hemorrhagic diathesis is unknown; but it is probably a toxin analogous to the hemorrhagins of snake venom.

4. In the other types of bleeding, arterial disease and hypertension are the chief factors.

5. The prognosis in all cases of nephritic hemorrhage is unfavorable, and generally hopeless when the hemorrhagic diathesis exists.

A CLINICAL STUDY OF FIVE HUNDRED CASES OF CONJUNCTIVITIS.[1]

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Conjunctivitis in its different forms constitutes one of the commonest diseases we have to deal with in ophthalmic practice. Its frequency varies according to race, climate, and environment; but when one considers how exposed the conjunctiva, a mucous membrane, is to external influences and how subject to involvement in nasal and postnasal inflammations, an average of 30 per cent. of all ophthalmic cases does not seem high. Conjunctivitis, too, more than any other disease of the eye, is treated by the general practitioner. In many cases he must treat it, so that I have thought it not out of place to report the results and lessons from a systematic examination of a series of cases.

In diseases of the eye, as in affections of other parts of the body, we look to etiology before prescribing or giving a prognosis. Our latest text-books still say: "As regards their etiology, much has yet to be learned, although considerable progress has been made in this direction within the past few years." It is quite true that much has yet to be learned, but enough is known today to enable us, by a bacteriological examination of the mucous or mucopurulent discharge, an important symptom of conjunctivitis, to obtain knowledge of much value in these directions. By using bacteriological methods we come to divide cases of conjunctivitis, not from their clinical features into catarrhal, purulent, and membranous with their numerous subdivisions, but rather into two groups: (1) Conjunctivitis with no known bacteriological cause, and (2) conjunctivitis in which there is a definite bacteriological factor.

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[1] Read at a meeting of the Montreal Medico-Chirurgical Society, June 7, 1907.

The first group includes such causes as exposure to winds, irritant gases, dust, eye-strain, rhinitis, inflammation of the lacrymal sac, smoke, gouty diathesis, trichiasis, alcoholism, etc. The second group includes the pathogenic microörganisms of the conjunctival sac, which are not few in number.

The method of finding these microörganisms is simple and quite within the reach of all. It consists in making a smear from the conjunctival discharge, staining, and examining it. Further examination of the conjunctival discharge is made by the inoculation of suitable culture media. In making a smear preparation considerable care is needed in obtaining a small particle of conjunctival secretion. Often the patients constantly wipe their eyes, so that it is almost impossible to obtain enough secretion for examination. Again, many cases of catarrhal conjunctivitis do not at certain times show any discharge. In either case, with a small platinum loop one may "pick out" from the inner canthus a loopful of conjunctival secretion. When this is smeared well over a glass slide and properly stained and examined, pathogenic microörganisms will be found, if present. Gram's method is by far the most satisfactory stain. It eliminates at once the frequently present Gram-positive bacilli, which in many instances are non-pathogenic. There are many little variations in doing Gram's stain, but that which I have found satisfactory is the one followed in Axenfeld's clinic: (1) Aniline-gentian violet, 25 seconds; (2) wash with water; (3) Gram's iodine solution, 15 seconds; (4) wash with alcohol until no blue comes away; (5) wash with water; and (6) counterstain with safranin, 5 per cent. solution, 5 seconds. Having become proficient in the staining, it is next necessary to become acquainted with the various microörganisms met with in both the healthy and the diseased conjunctival sac.

A knowledge of the pathogenic microörganisms can be obtained only by seeing a large number of cases of conjunctivitis. While much satisfactory work can be done by a study of smear preparations, the real differentiation of the organisms must come from a study of their cultural characteristics—the especial difficulty of conjunctival bacteriology. The bacteria pathogenic for the conjunctival sac require special media in nearly every case. Even on special media the growths are usually sparse and likely to be overgrown by more easily cultivated organisms.

But what are the commonest pathogenic microörganisms met with in the different forms of conjunctivitis? In the Freiburg clinic, during 1902 to 1905 inclusive, 900 cases of acute and chronic conjunctivitis were examined, with the following results:

Morax-Axenfeld diplobacillus 519 cases; Koch-Weeks bacillus 41 cases; pneumococcus 34 cases; gonococcus 12 cases; Loeffler's bacillus 6 cases; streptococcus 5 cases; Friedländer's bacillus 2 cases, and Pfeiffer's bacillus 3 cases.

Gonin, in the clinic at Lausanne, examined 310 cases of catarrhal conjunctivitis with the following results: Morax-Axenfeld diplobacillus 185 cases; Koch-Weeks bacillus 10 cases; pneumococcus 10 cases; streptococcus 5 cases, and staphylococcus 83 cases.

Pollock, of Glasgow, examined 361 cases of conjunctivitis with the following results: Koch-Weeks bacillus 189 cases; Morax-Axenfeld diplobacillus 62 cases; pneumococcus 9 cases; pneumococcus and diplobacilli 3 cases; gonococcus 17 cases; gonococcus and other organisms 3 cases; staphylococci and streptococci 15 cases; Koch-Weeks bacillus and Bacillus subtilis 1 case, and negative 62 cases.

Morax, of Paris, reports as follows: Koch-Weeks bacillus 175 cases; Morax-Axenfeld diplobacillus 258 cases; gonococcus 121 cases; pneumococcus 18 cases; influenza bacillus 6 cases; Loefler's bacillus 2 cases; staphylococci 6 cases, and unknown 10 cases.

Lundsgaard, of Copenhagen, examined 107 cases with the following results: Morax-Axenfeld diplobacillus 36 cases; pneumococcus 15 cases; gonococcus 5 cases; Koch-Weeks bacillus 3 cases; staphylococcus 5 cases; diphtheria bacillus 1 case, and Bacillus coli 1 case.

Bach and Neumann report on 110 cases as follows: Morax-Axenfeld diplobacillus 35 cases, pneumococcus 15 cases, and staphylococcus 53 cases.

Veasey in 64 cases of acute catarrhal conjunctivitis found the pneumococcus in 53 cases and the Koch-Weeks bacillus in 3 cases.

My report consists of a study of 500 cases, of which the results are as follows: Morax-Axenfeld diplobacillus 200 cases; pneumococcus 13 cases; gonococcus 10 cases; Koch-Weeks bacillus 7 cases; influenza bacillus 3 cases; meningococcus 1 case; McKee bacillus 9 cases; Micrococcus catarrhalis 12 cases; streptococcus 24 cases; staphylococcus 50 cases; Bacillus coli 5 cases; Bacillus xerosis, Bacillus Hoffman, and saprophytes 102 cases, and negative results 64 cases.

From these figures it will be seen that the most common forms are the Morax-Axenfeld, the Koch-Weeks, the gonococcic, and the pneumococcic conjunctivitis. It is an interesting fact that in different countries and localities different forms of conjunctivitis seem to predominate. In Nebraska and Washington pneumococcic conjunctivitis is the prevalent form; in New York Koch-Weeks is most frequently seen; while in Montreal by far the most common form is the Morax-Axenfeld.

In view of the impossibility of discussing the many interesting features of all forms, I have thought it best to consider a few of the commonest forms seen in Montreal, dwelling especially upon the two most important forms, the Morax-Axenfeld and the gonococcic infections, and a third form of conjunctivitis which I believe has not yet been described.

GONOCOCCIC CONJUNCTIVITIS. The position of the gonococcus in purulent ophthalmia unfortunately has not changed much. We all know too well what progress it makes if once allowed entrance to the conjunctival sac, and what efforts are needed to stay its course. While I have nothing new to offer on gonococcic infections, which seem as virulent today as ever, we have learned or are learning much about purulent ophthalmia, both in the infant and adult. Let me illustrate with some cases.

CASE I.—*Ophthalmia neonatorum; typical history and clinical picture.* Smears from the conjunctival discharge showed numerous Gram-negative intracellular diplococci. This was thought to be an exceedingly virulent infection, but when the patient was brought back three days later the condition had almost entirely subsided. The inoculated tubes explained it. The organisms seen in the smear preparations were not gonococci, but Micrococcus catarrhalis or pseudogonococcus.

CASE II.—*Ophthalmia neonatorum: typical history and clinical picture.* Repeated examination of the pus gave negative results. From cultures were obtained only a few colonies of the staphylococcus. The patient had been seen on Saturday, and when the mother returned the next Saturday she told me the baby's eyes had received no treatment since the Tuesday before.

CASE III.—*Ophthalmia neonatorum: typical history and clinical picture.* From numerous smears made no microörganisms were found. Cultures, however, gave pure growths of the gonococcus. The case ran a stubborn course entirely different from Cases I and II.

CASES V, VI, VII were all purulent ophthalmias in adults. The clinical pictures were typically gonorrheal infections. The smears in each case showed the pus cells filled with Gram-negative diplococci. Tubes in each case showed the organisms to be the pseudogonococcus. The course in each case was mild, and of three or four days' duration.

The question of Gram-negative diplococci in the conjunctival sac is one to which I have given some thought. Why does one case of supposed gonorrheal ophthalmia run such a mild course and the next such a severe one, when from each Gram-negative diplococci are obtained? Different virulence of the gonococcus we say; granted, but also different microörganisms. I believe many cases of ophthalmia neonatorum and purulent conjunctivitis put down to gonococcic infection are not of that origin, but, as in the cases here cited, are due to other Gram-negative diplococci of much milder virulence.

KOCH-WEEKS CONJUNCTIVITIS. This is a form of conjunctivitis first reported by Weeks, in 1887, and since then reported almost universally. The Koch-Weeks bacillus is now recognized as the most common cause of acute contagious conjunctivitis. This is a form which clinically may begin as an acute catarrhal conjunctivitis and run a mild course of a few days, or it may become very severe, with œdema of the bulbar conjunctiva and lids, and thick, purulent discharge. It attacks children and adults alike, tends to appear in the spring and fall of the year, and is one of the most contagious diseases we have to deal with. Happily complications seldom occur in this form; the most common are ulceration of the cornea and iritis. Pneumococcic conjunctivitis is a form which seems to favor northern climates, occurs especially in the winter, and is often associated with coryza. The clinical picture is a varying one from a mild catarrhal conjunctivitis to marked red-rose œdema, swelling of the lids, hemorrhages in the upper lid, and profuse discharge. It seems to attack children preferably, and is likely to be complicated by iritis, which may last longer than the conjunctivitis. Müller first described a bacillus which he found in cases of trachoma which he did not recognize as Bacillus influenzæ. Zur Nedden later reported cases of blennorrhœa neonatorum and other cases of acute conjunctivitis from which he isolated a bacillus which he called the pseudo-influenza bacillus. Numerous writers have reported influenzal conjunctivitis, an acute catarrhal conjunctivitis which may accompany systemic influenza.

MORAX-AXENFELD'S CONJUNCTIVITIS. The examination of two hundred cases of Morax-Axenfeld conjunctivitis here has brought out some interesting points, and what I have to say on this subject is based entirely upon the cases seen here. Described first by Morax and Axenfeld, and called by the latter because of its persistent qualities chronic diplobacillary conjunctivitis, this form has since been reported from many lands. Its presence in Canada was reported first in 1905 by me, and it has not as yet been reported here by any other writer. It is today one of the best known and most frequently seen diseases of the conjunctival sac. This form of conjunctivitis was first described as a chronic catarrhal conjunctivitis, which from the reddening seen at the outer and inner canthi was sometimes called angular conjunctivitis, and at times, from the associated blepharitis, blepharoconjunctivitis.

It is seen generally in one of four forms: (1) A mild catarrhal conjunctivitis with little or no involvement of the bulbar conjunctiva. (2) A catarrhal conjunctivitis with reddening at the outer and inner canthi. (3) A severe catarrhal conjunctivitis with marked involvement of the bulbar and palpebral conjunctiva and marked blepharitis. (4) An acute purulent conjunctivitis.

I have been struck with the number of acute cases that we have seen here—cases which clinically were Koch-Weeks or pneumococcic conjunctivitis, in which examination of the discharge showed them to be diplobacillary. Cases have also been seen in which clinically the disease was follicular conjunctivitis, as well as other cases associated with trachoma and phlyctenular conjunctivitis. The cases associated with trachoma, contrary to the results of Myerhoff, did well on instillations of zinc sulphate. This, more than any other form of conjunctivitis, can be diagnosticated clinically; certain types are easily recognized, but on the other hand no form differs so much in its clinical appearance as does this. Morax-Axenfeld conjunctivitis differs here in many ways from the descriptions in the literature:

1. Many of these patients had severe subjective symptoms. They complained of pain in the eyes which was worse at night.
2. Many of the cases were acute, simulating blennorrhœa.
3. This form has been seen here very often during cold and wintry weather. I noted a number of times that after cold weather and snow a series of patients would present themselves at the clinic. The last time noted was April 10, the day after a snow-storm and wintry weather.
4. The cases associated with trachoma reacted well to the zinc sulphate.
5. Seven cases complicated with ulceration of the cornea were cured with instillations of zinc.
6. A series of cases of phlyctenular conjunctivitis in adults complicated by diplobacillary infections were seen.
7. Diplobacilli were found constantly in the noses of patients suffering from this form of conjunctivitis.
8. One patient, a boy was seen with excoriation about the nostrils; the examination of a little secretion showed the presence of diplobacilli.
9. In no case of diplobacillary conjunctivitis have I seen zinc sulphate fail. I have treated cases with silver nitrate and argyrol to no purpose. I have seen cases occurring in children return without improvement, but here the failure was not due to the zinc, but due to the inability to instil the drops. I have treated with argyrol or silver nitrate cases of trachoma in which diplobacilli were found in the conjunctival sac, but I never noticed any improvement in these cases until I began instilling zinc sulphate.

In addition to these we have seen a few cases that are of interest more from their rarity than from their practical value. During the past year we have had a number of cases of epidemic cerebrospinal meningitis. Conjunctivitis is a common complication of this disease. Of this number two cases had definite acute simple conjunctivitis. From Case I smears showed Gram-negative intracellular diplococci; cultures on hemoglobin agar gave a growth of Gram-negative diplococci, which were meningococci, that is, meningococcic conjunctivitis. Another case was one in which Petit's diplobacillus was present in the conjunctival sac. The conjunctiva had none of the clinical signs of diplobacillary infection. The diplobacillus was differentiated from Morax-Axenfeld's by its cultural features. During the winter we had an epidemic of streptococcic conjunctivitis. The cases were all in infants. The clinical picture was one of pseudomembranous conjunctivitis. The infection was mild, reacting quickly to treatment. Another rare form seen was a case of lacrymal conjunctivitis of Parinaud. The patient, an adult male, came with a severe streptococcic conjunctivitis with severe pain in the eye, swelling of the lids, and involvement of the pre-auricular gland. The day following an iritis (absorption or toxin iritis) was present. The case ran a severe course

McKEE CONJUNCTIVITIS. Upon April 6, 1907, a mother brought her infant of nine months to the Montreal General Hospital, and complained that the baby's eyes had been sore and running for five days. The clinical picture was as follows: Both eyes were involved. Upon the lashes was much dried secretion. The palpebral conjunctiva was intensely congested, while the bulbar conjunctiva was quite normal; the lids were not red or swollen. From the conjunctival sac there was a profuse mucopurulent discharge freely mixed with tears. A little of the discharge was smeared well over a glass slide, fixed, and stained. Upon examining the slide I saw here and there tiny Gram-negative bacilli which seemed too short and thick for the Koch-Weeks bacillus and too thick for Bacillus influenzæ. I had seen before this same bacillus, but in the hurry of a large outdoor clinic I had not pursued it farther, contenting myself with the thought that it was Bacillus influenzæ.

Tubes of blood serum, plain agar, bouillon, and hemoglobin agar were then inoculated and put in the incubator. After twenty-four hours a growth was seen upon the hemoglobin agar. The other tubes were negative. Smears showed this same small Gram-negative bacillus with the staphylococcus. The bacillus after a few days was obtained in pure culture, conjunctivitis set up, and from the conjunctival discharge this same small bacillus was obtained.

During a month nine such cases have presented themselves at the outdoor department. They have all been infants and all from among the Jewish colony here. They have all shown definite characteristic clinical features and from each case this same bacillus has been isolated.

Cultural Features. The tubes of hemoglobin agar inoculated with some of the conjunctival discharge gave a pretty and characteristic result which was constant in all the cases. After twenty-four hours in the incubator one sees over the surface numerous separate colonies of Staphylococcus albus.

Around each of these separate colonies radiating from them as centres are seen very fine, colorless, shiny, rounded colonies. These colonies are exceedingly fine, seem to keep separate, and extend over the whole surface. The original cultures from all the cases were as the one described and showed how well this bacillus grows in commensal relationship with staphylococci. From the original tube the bacillus was obtained in pure culture by surface seeding a plate of hemoglobin agar. In trying to obtain it in pure culture by transferring from tube to tube the growths were always contaminated with staphylococcus. Over a plate of hemoglobin agar, however, by smearing a loop inoculated from one of the small colonies as far from the albus as possible, the bacillus in pure culture was readily obtained. The growth of the bacillus on hemoglobin agar is characteristic. The slant will be seen covered with a mass of tiny colonies. They remain separate and distinct, are colorless and, as will be seen from the tubes, the colonies are no larger than the sharp end of a pin; indeed the growth, though profuse, is at times exceedingly difficult to recognize. It is seen much better by artificial light than by daylight, especially with the proper reflection of light. The growth of the bacillus is very easily affected, even on hemoglobin agar, upon which it grows best. Upon freshly made hemoglobin agar it will not grow in less than forty-eight hours. The reaction of the media, too, is of the greatest importance. Hemoglobin agar neutral, 1 alkaline, 0.65 acid, 1 acid, and 1.75 acid to phenolphthalein was made up. Upon alkaline media it would not grow; upon the neutral and 0.65 acid it was transferable with great difficulty. The most satisfactory reaction and the one which has been latterly used is the 1 and 1.75 acid. The cultures on hemoglobin agar remain viable from two to three weeks, provided there is sufficient water of condensation in the tubes. One culture inoculated on April 11 was transferable on April 25, but not on May 3. It had not been kept in the cold nor had special care been taken of it.

For the human conjunctiva this bacillus is pathogenic. A conjunctival sac which was found to be free from microörganisms was well flushed out with warm water. Fifteen minutes later the sac was inoculated with a loopful of a twenty-four-hour growth. Twelve hours later there was a feeling of "something in the eye" and six hours later the eye showed a marked catarrhal conjunctivitis. The palpebral conjunctiva of the lower lid was especially congested. The posterior conjunctival vessels were somewhat dilated, otherwise the bulbar conjunctiva was not involved. From the mucopurulent discharge, which was here, too, freely mixed with tears, the organism was obtained.

With the washing from a slant on hemoglobin agar an ordinary house mouse was inoculated intraperitoneally. Eighteen hours later the mouse was unable to move and six hours later was dead. At autopsy the peritoneum was found injected and filled with a gelatinous, sticky exudate. Smears showed the pus cells filled with the bacilli, with little or no fibrin. Inoculations from the peritoneum on hemoglobin agar gave pure growths of the bacillus. This tube was washed down and inoculated intraperitoneally into a white mouse, and twenty hours later the mouse was dead. Postmortem a marked peritonitis of the gelatinous type, similar to that of Case 1, was found. From the peritoneum and from the heart's blood the bacillus was obtained in pure culture.

This microörganism, then, a cause of conjunctivitis, needs to be differentiated from the other known bacteriological causes of conjunctivitis. Differentiation from Morax-Axenfeld's diplobacillus, Petit's diplobacillus, Friedländer's bacillus, Bacillus coli, and Bacillus subtilis may be dealt with in a few words. It is far too different morphologically and culturally to necessitate going into minutiæ. The pathogenic microörganisms of the conjunctival sac from which it must be differentiated, however, are the Koch-Weeks bacillus and the different influenza bacilli, the influenza bacillus of Pfeiffer, the so-called influenza bacillus of conjunctivitis of Müller, and the pseudo-influenza bacillus of Zur Nedden.

1. *Clinical Considerations.* While the different bacteriological factors causing conjunctivitis do not always, yet in the majority of cases these organisms do, give rise to well-marked characteristic pictures. In Koch-Weeks conjunctivitis one finds reddened and œdematous lids, intense injection of the bulbar conjunctiva, with profuse mucopurulent discharge. In influenzal conjunctivitis there is a well-marked involvement of the bulbar conjunctiva and signs of influenza elsewhere. Bacillus influenzæ has been found in the normal conjunctival sac and in odd cases of conjunctivitis in which there were no general signs of influenza, but in the majority of cases influenzal conjunctivitis accompanies systemic influenza. Clinically, Koch-Weeks', influenzal, and the form of conjunctivitis set up by the bacillus here described, are very different.

2. *Morphology.* Differentiation by morphology is unsatisfactory and unnecessary. While the Koch-Weeks bacillus may be differentiated from Bacillus influenzæ by its appearance on the slide, and while the bacillus here described seems shorter and thicker than Bacillus influenzæ, I am ready to admit that morphologically they are strikingly similar.

3. *Cultural Features.* On media these organisms differ widely. The Koch-Weeks bacillus does not grow well on hemoglobin agar, as here used. It grows best on hydrocele or ascitic agar; when growing with Bacillus xerosis it is hardly perceptible and dies out very quickly in forty-eight hours or a little longer. Bacillus influenzæ in the presence of hemoglobin, after twenty-four hours, shows a profuse raised growth of round, whitish colonies. The growth is easily seen, the colonies being the size of an ordinary pinhead; older colonies show some change in color. The bacillus here described, on hemoglobin-agar gives a very characteristic appearance. Over the surface of the agar, with a proper reflection of light is seen a mass of tiny, pinpoint, colorless colonies. The growth is not easily

seen. This organism has also been cultivated on media free from hemoglobin.

4. *Pathogenicity.* The Koch-Weeks bacillus, while pathogenic for the human conjunctival sac, is not pathogenic for animals. Bacillus influenzæ is slightly pathogenic for some animals. Pfeiffer found, by injecting Bacillus influenzæ intravenously into rabbits, that a characteristic effect was produced. He attributed the results to toxic products present in the culture. In none of his experiments was he able to obtain effects resembling septicemic infection. Cantani by injecting Bacillus influenzæ into the anterior portion of the brain of rabbits succeeded in producing an acute encephalitis; the bacilli, however, were never found in the blood or other organs. This bacillus is pathogenic for mice. In every case in which it was injected intraperitoneally marked peritonitis was set up, and a septicemia, as evidenced by pure cultures of the bacillus being obtained from the heart's blood, resulted. This organism then is similar to the different influenza bacilli in morphology and in its growing well in commensal relationship with the staphylococcus. It is not Bacillus influenzæ for the following reasons: (1) The form of conjunctivitis set up differs from influenzal conjuncivitis. (2) This bacillus on hemoglobin agar differs widely from Bacillus influenzæ. On this media under most favorable conditions the growth is hardly perceptible and differs from Bacillus influenzæ on the same media, in size, color, and appearance. (3) This bacillus has been cultivated on media free from hemoglobin. (4) It is viable for a much longer period than Bacillus influenzæ. (5) It is pathogenic for the human conjunctiva.

We have then to deal with a new form of conjunctivitis set up by a new etiological factor. That factor resembles somewhat the bacillus of influenza, but differs from it widely on media, viability, and pathogenicity. In the examination of these five hundred cases of conjunctivitis, this series occurring within a month seems to be different from previously described types. Nine cases constitute a small number from which to draw conclusions, but the characteristic clinical features of these cases, caused by a microörganism not as yet described as a pathological factor in conjunctivitis, leads me to the conclusion that we have here to deal with a new form of conjunctivitis set up by a new microörganism of the conjunctival sac.

Until the clinical features of this disease from which the bacillus should take its name are better understood, I shall call it Bacillus McKee.

Treatment. Bacteriological methods, since they reveal to us the cause of conjunctivitis, also suggest the treatment. In mild cases of catarrhal conjunctivitis in which no microörganisms are found, the refraction and muscular balance of the eyes should be examined, nasal diseases should be attended to, and when there is any indication for it systemic treatment should be instituted.

Koch-Weeks conjunctivitis calls for preventive measures, the isolation of the patient, and, if the epidemic is a large one, closing of schools and such institutions. For Koch-Weeks conjunctivitis the application of the silver preparations has been the most satisfactory. Morax noted a great diminution in the number of bacilli after the application of silver nitrate. With this, cold compresses are found very beneficial. When there is any tendency to ulceration of the cornea or iritis, preventive measures need to be adopted immediately. Pneumococcic conjunctivitis, which is usually accompanied by severe swelling of the lids, is best treated with cold compresses and one of the silver preparations.

In gonococcic ophthalmia silver nitrate has been used extensively. Personally I prefer argyrol. In a limited number of cases I have never seen it fail.

In Morax-Axenfeld conjunctivitis we have in zinc sulphate a specific. Many cases which present the classical clinical features of Morax-Axenfeld blepharoconjunctivitis will be found to be not due to that microörganism, but to errors of refraction or other causes.

Bacteriological methods will never excuse one for neglecting such etiological factors as these, which should ever be borne in mind. Constant reference of these factors with no attempt at bacteriological methods is equally as inexcusable. Cases of acute catarrhal conjunctivitis occurring in children, especially in the spring, are probably not due to rhinitis, and while referring cases to the rhinologist is by no means to be discredited, certainly it is not the first procedure in such cases. Cases of one-sided conjunctivitis associated with some lacrymal disturbance due primarily to nasal affections are different, and here of course nasal treatment is the first indication.

Some Clinical Considerations. The scientific value of conjunctival bacteriology is acknowledged, but the practical value of this proceeding has not been given the position which it merits. This work is especially useful to the clinician, because so much of it can be done along with his clinical work in his office. These methods teach us that one must not put down all cases of acute simple conjunctivitis to Koch-Weeks infection, all cases of chronic catarrhal conjunctivitis to Morax-Axenfeld's diplobacillus, membranous and pseudomembranous forms to the diphtheria bacillus, and all purulent cases to gonococcic infection. In acute simple conjunctivitis we may find many other organisms present, in chronic catarrhal conjunctivitis none at all, in purulent ophthalmia many others than the gonococcus; indeed, the gonococcus is only one of eight or ten Gram-negative diplococci which have been found in diseased conditions of the conjunctival sac.

All these forms may be very similar in clinical features. The different bacteria cause at times definite characteristic forms of conjunctivitis, but at other times the clinical pictures give one no knowledge of the etiological factors, so that the definite diagnosis depends upon the microscopic examination.

Bacteriological methods are of great assistance in different ways. Many cases of conjunctivitis are thus rapidly diagnosticated. We see many cases of trachoma which have received treatment for some weeks elsewhere. The similarity in their clinical picture to diplobacillary conjunctivitis would surprise one, the reddening at the outer and the inner canthi being at times marked. The presence of diplobacilli in the smear preparation settles the diagnosis at once. Negative results assist in excluding other forms of conjunctivitis and coming to the diagnosis of trachoma. The value of this knowledge will be better appreciated when one tries to cure trachoma with zinc, and diplobacillery conjunctivitis with silver. The results in the one case will be as unsatisfactory as in the other.

In ophthalmia neonatorum the knowledge that one is dealing with Bacillus coli or Micrococcus catarrhalis rather than with the virulent gonococcus or pneumococcus or the easily handled Morax-Axenfeld diplobacillus is of the greatest service. In purulent conjunctivitis it is important to know which of these many organisms is active. All this knowledge is of importance because when one has the gonococcus to deal with he knows he has a severe infection, but when the purulent ophthalmia is not due to the gonococcus or the pneumococcus the disease will run a mild course. The Koch-Weeks or Morax-Axenfeld infections require different methods of treatment, as also do those cases in which the bacteriological examination is negative. Clinical conditions will not give one this knowledge; to get such specific information one must turn to bacteriological methods. I do not at all wish to go to extremes, or to put bacteriological methods and examinations before clinical work; far from it, since clinical work in conjunctivitis has gone on and could go on perhaps without bacteriology. But I wish to put bacteriological methods and clinical work together. Together they make the diagnosis easy and sure, and when one has diagnosticated he knows.

Koch-Weeks conjunctivitis is the most contagious disease we have to deal with in ophthalmic practice, a disease which spreads rapidly, increasing in severity as it goes. In gonococcic as in pneumococcic and streptococcic infections we have virulent organisms to deal with which require constant care on the surgeon's part, while in purulent conditions from such microörganisms as Bacillus coli, Micrococcus catarrhalis, or staphylococcus we have comparatively mild infections. In all these cases treatment and results vary as do the etiological factor. Diplobacillary infection is a form of conjunctivitis which with proper treatment is easily cured, but without which it is a disease most chronic and likely to be complicated with ulceration of the cornea—resembling in severity the pneumococcic ulcus serpens. Even in phlyctenular conjunctivitis, these methods will repay one. In trachoma negative results have their value. In many other cases, too, negative results tell us to look to other factors in etiology and suggest other lines of treatment. In all these forms bacteriological methods give us our diagnosis and, by telling us which of these different factors we are dealing with, put preventive measures, treatment, and prognosis on a practical basis.

This is what bacteriology does for the clinician, not only in conjunctival but in corneal work as well; this is where bacteriological methods and clinical considerations meet. Can one doubt their practical value?

The hemoglobin agar and other special media were made under Dr. Duval's direction. In working out Bacillus McKee he assisted me in many ways. I take great pleasure in here recording my indebtedness and thanks.

THE OPSONIC INDEX IN ERYSIPELAS AND ITS RELATION TO TREATMENT BY INOCULATION OF KILLED STREPTOCOCCI.

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After the observations of Wright and Douglas had stimulated investigations of all diseases of infectious origin, Dr. James C. Ayer kindly placed at our disposal for the determination of opsonic indices and for vaccination all cases of erysipelas treated in the wards of Bellevue Hospital, from January 1 to February 15 of this year.

During this time about 100 cases of erysipelas were admitted. Many of these cases were not suitable for investigation, as the patients entered the hospital at a late stage of the disease, after desquamation had occurred, or after there was complication with other diseases. Those cases in which no definite diagnosis of erysipelas could be made out were not studied. Altogether, the opsonic index was studied in 36 patients, and 37 patients received one or more injections of killed cultures.

The etiology of erysipelas was well established by the work of Fehleisen, who regarded Streptococcus erysipelatos as the cause of the disease. It is probable that streptococci causing erysipelas do not differ from those causing wound infections, for erysipelas often follows wound infections—infections of the mouth and scarlet fever. Moreover, streptococci from erysipelas transferred to other individuals may cause puerperal fever and other infections. In our series, erysipelas frequently followed coryza and wound infections.

Streptococci were isolated from cases of cellulitis and from blebs upon the skin in cases of erysipelas. Although it is possible that infection with streptococci can be best treated with killed cultures isolated from the case in question, the same culture being used for the determination of the opsonic index, yet the course of the average case of erysipelas makes it impracticable to isolate cultures from every case of the disease and to make a vaccine for its treatment. In many cases no blebs are formed, and even if a bleb were artificially produced, the patient might have recovered before the culture could have been isolated and the vaccine made. Moreover, since most patients are admitted to the hospital during the acute or during the advanced stage of the disease, treatment to be of service must be administered without delay.

A uniform mixture of streptococci from 4 cases of erysipelas was used in determining opsonic indices and in making vaccines. One of these cultures came from a bleb present with facial erysipelas and another was obtained from the discharging ear of a patient with erysipelas of the ear and otitis media; the third culture was from a case of erysipelas affecting the leg, and a fourth was from a bleb with erysipelas of the face and scalp. All of these microorganisms had the same cultural characteristics, and grew in long chains in bouillon. They produced hemolysis, caused acid formation in milk, and did not ferment inulin.

Technique of Determining the Opsonic Index. One tube of each of the first two cultures and two tubes of each of the third and fourth cultures were incubated twenty-four hours on glycerin-glucose-agar, to which 20 per cent. of sterile sheep serum had been added. From 2 to 3 c.c. of sterile salt solution was used to prepare an emulsion from these six slant agar growths. The emulsion was put in a small test tube containing some sterile sea sand; the tube was sealed in the flame and kept during one and a half hours in the shaking machine. The sand and emulsion in the tube were centrifuged for one minute and the fluid was drawn off. In this way, short chains of from two to four cocci were uniformly obtained.

The serum used for control was a mixture from two healthy adults. Our leukocytic emulsion was obtained by drawing ten drops of blood into about 10 c.c. of 1.5 per cent. potassium citrate in normal salt solution. The mixture was centrifuged, and without further washing the leukocytes were drawn off.

The mixture of leukocytes, bacteria, and serum in capillary tubes was incubated in the opsonizer of Wright at 37° C. for twenty minutes. The slides were fixed with methyl alcohol and stained with Loeffler's methylene blue. The cocci in fifty definitely outlined and isolated polynuclear leukocytes along the upper and low borders of the spread were counted. The slides uniformly showed well-preserved leukocytes and the chains of cocci were well broken up.

This method of determining the opsonic index does not differ essentially from the one used by Wright and Douglas. A series of experiments was made to determine the accuracy of the method. In these tests, Staphylococcus aureus, of which suspensions are easily prepared, was used instead of Streptococcus erysipelatos. The attempt was made to determine (1) the limits of error when several specimens of serum are drawn almost simultaneously from the same individual, and (2) the variation of the opsonic power of healthy individuals.

I. Five specimens of the blood of a healthy adult were drawn from five fingers of the same hand. The leukocytes used for the determination were washed twice in 1.5 per cent. potassium citrate in normal salt solution.

TABLE I.

	1st 100 leukocytes counted.	2d 100 leukocytes counted.	Phagocytic index.
Specimen 1	166	196	1.810
" 2	176	183	1.795
" 3	198	199	1.985
" 4	185	175	1.800
" 5	230	190	2.100

II. A series of ten specimens of blood from ten healthy adults was studied to determine the variations in opsonic power in healthy individuals. The leukocytes were twice washed with 0.85 per cent. salt solution. The number of cocci taken up by one hundred polynuclear leukocytes was counted in two preparations separately incubated, and counted by two workers, A and B. The phagocytic indices are as follows:

TABLE II.

Source.	A	B
N.	9.48	7.63
J.	9.16	5.20
B.	8.75	6.80
McL.	7.27	4.37
Z.	6.63	5.12
C.	6.60	5.37
S.	5.89	4.82
O.	5.81	4.57
H.	5.49	6.03
Ns.	1.91	1.37

In the early observations of Wright and Douglas, the opsonic index was determined by comparing the serum to be tested with a mixture of several normal sera (pooled serum). This practice has been almost universally discarded, comparison being made with a single serum. The great error introduced by this method is shown by tables III and IV, which contain the indices obtained by using each serum for comparison with all of the others. Table III (Series A) shows that the normal opsonic index varies from 0.52 to 1.93, and in Table IV (Series B) it varies from 0.57 to 1.75.

TABLE III—A.

Denominator.	Numerator. I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.
I.	...	0.97	0.86	0.77	0.70	0.70	0.62	0.62	0.58	0.52
II.	1.03	...	0.89	0.79	0.72	0.72	0.64	0.64	0.60	0.52
III.	1.15	1.12	...	0.89	0.81	0.80	0.72	0.72	0.67	0.60
IV.	1.30	1.26	1.12	...	0.91	0.90	0.81	0.80	0.76	0.68
V.	1.42	1.38	1.23	1.10	...	1.00	0.89	0.88	0.83	0.74
VI.	1.42	1.39	1.23	1.10	1.03	...	0.89	0.88	0.83	0.74
VII.	1.61	1.56	1.38	1.23	1.13	1.12	...	0.99	0.93	0.83
VIII.	1.62	1.57	1.39	1.24	1.13	1.13	1.01	...	0.94	0.84
IX.	1.73	1.67	1.48	1.32	1.21	1.20	1.07	1.06	...	0.89
X.	1.93	1.87	1.66	1.48	1.33	1.32	1.20	1.19	1.10	...

TABLE IV—B.

Denominator.	Numerator. I.	III.	IX.	VI.	II.	V.	VII.	VIII.	X.	IV.
I.	...	0.89	0.79	0.70	0.68	0.67	0.63	0.60	0.57	0.57
III.	1.12	...	0.89	0.79	0.76	0.75	0.71	0.67	0.64	0.64
IX.	1.27	1.13	...	0.89	0.86	0.85	0.80	0.76	0.72	0.72
VI.	1.42	1.27	1.12	...	0.97	0.95	0.90	0.85	0.81	0.81
II.	1.47	1.31	1.14	1.02	...	0.98	0.93	0.88	0.84	0.84
V.	1.49	1.33	1.17	1.05	1.02	...	0.94	0.89	0.85	0.85
VII.	1.59	1.41	1.25	1.11	1.08	1.06	...	0.95	0.91	0.91
VIII.	1.67	1.49	1.21	1.18	1.14	1.12	1.05	...	0.96	0.96
X.	1.75	1.56	1.38	1.23	1.19	1.17	1.10	1.05	...	1.00
IV.	1.75	1.56	1.38	1.23	1.19	1.17	1.10	1.05	1.00	...

III. Blood was taken from five healthy individuals and a pool was made by mixing equal volumes of each of their sera. The phagocytic indices were determined in duplicate after separately incubating and counting the opsonic preparation, and an average of these determinations was compared with the phagocytic index obtained with the pooled serum. The phagocytic indices determined as the result of counting 100 polynuclear leukocytes in each preparation were as follows:

	Z.	N.	J.	McL.	S.	Pool.	Calculated average.
A	6.08	7.64	5.61	5.88	7.25	6.49	6.57
B	6.89	7.79	7.78	7.11	7.17	7.20	7.75

IV. This experiment was made with blood from five individuals, one of whom suffered with a streptococcic and staphylococcic infection, and another with a staphylococcic infection. A pool was made of the five sera and the phagocytic index determined by counting the cocci in 50 polynuclear leukocytes.

	B	C
1. Normal	2.72	3.004
2. Normal	2.24	3.160
3. Normal	3.06	3.960
4. Streptococcic and staphylococcic infections	2.08	3.100
5. Staphylococcus aureus infection	3.40	4.080
6. Pool of five sera	2.80	3.300
Calculated pool	2.70	3.460

It is evident that the method of determining the opsonic index usually employed is not accurate. There is not always agreement between results obtained by counting 100 and 200 polynuclear leukocytes on the same slide. When separate specimens of serum from the same individual are made, there is even greater error. When sera from different healthy individuals are tested by comparison with a single normal individual, such great error is introduced that the determination has little value. The preceding tests show, moreover, that comparison of the serum to be tested with a pooled serum decreases this range of error.

Observations upon the opsonic index were undertaken with the methods recommended by Wright and Douglas, and subsequently employed almost universally. The error introduced by comparison with a single serum was diminished by the use of a mixture of two normal sera. To further diminish the errors which occur with all determinations of the opsonic index, composite charts embodying a considerable number of observations have been prepared, when possible. The possible error introduced by collecting observations made upon a variety of cases, some of which were complicated by conditions other than erysipelas, is recognized.

VACCINE. Twenty-four-hour cultures of the same strains of streptococci used for testing the opsonic index were used to make the vaccine, which was standardized by estimating the number of cocci per cubic centimeter.

OPSONIC INDEX IN ERYSIPELAS. Before determining the changes in opsonic index produced by vaccination, a number of unvaccinated cases were studied. The most instructive of these cases are the two which follow.

CASE I.—S., a man, aged thirty-eight years, who had a migratory, recurrent erysipelas of the face, ears, scalp, and neck, was admitted to the hospital on the fourth day of his disease. When his face was involved, his index was 0.7; when it was desquamating, the index had risen to 1.3; when his face was again involved, seven days later, the index was 1.4; and when it was desquamating again, the index had dropped to 0.8. When his neck became involved, one day after his face began to desquamate for the second time his index was 1.0; two days later, with an index of 1.1, his back became involved, and three days later, with an index of 3.2, his shoulders and neck were again involved, and two days later, with an index of 1.1, the back and shoulders began to desquamate. (Chart I.)

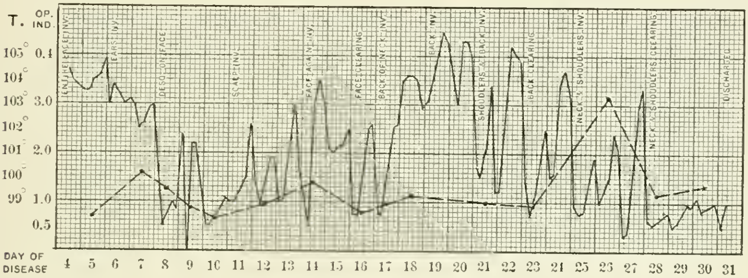

Chart I.—Temperature (unbroken line) and opsonic index (broken line) in a case of erysipelas (Case I).

Case II.—A., a man, aged twenty-four years with erysipelas of the face and ears, was admitted on the eighth day of the disease with an index of 0.9. The next day his temperature dropped and desquamation began; his index was found to be 1.1. Subsequently the index, though the patient was perfectly well, remained below unity. (Chart II.)

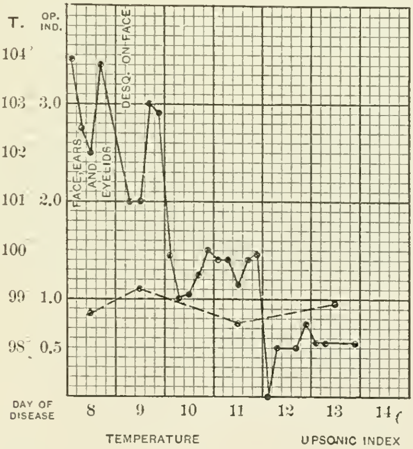

Chart II.—Temperature (unbroken line) and opsonic index (broken line) in a case of erysipelas (Case II).

In these two cases there has been no constant change of index corresponding with desquamation and recovery.

To further study the relation of the opsonic index to the course of the disease, the indices of all patients who had received no vaccine were tabulated (Table V) with regard to the day of the disease, and an average opsonic index for each day was determined.

TABLE V.

TABLE SHOWING THE OPSONIC INDEX AT THE TIME OF ADMISSION, BEFORE KILLED CULTURES WERE INJECTED, AND THE DAY OF THE DISEASE.

Day of disease.

1	2	3	4	5	6	7	8	9	10	11
0.6	1.2	0.6	0.7	0.7	0.4	0.9	0.8	1.1	0.6	1.0
0.3	1.1	1.1	1.1	1.0		1.1	0.8	0.9		0.7
	1.0	2.4	1.4	0.9		0.6	1.3	1.7		1.0
		1.7	1.5	0.6		1.6				
			0.7	1.1						
			1.0							
			1.2							
0.45	1.1	1.45	1.1	0.86	0.4	1.05	0.96	1.23	0.6	0.9

Average.

The composite chart indicates that erysipelas causes an increase of the opsonic index which reaches its maximum about the third day of the disease, and is followed by a gradual fall. The subsequent course of the chart represents, in very large part, observations made upon recurrent, migratory, and complicated cases. Ruediger[1] found that the index was high during an attack of erysipelas.

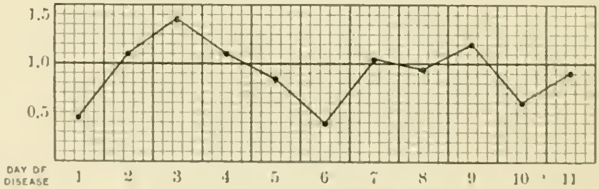

CHART III. To show the average opsonic index and the day of disease before killed cultures of streptococci were injected.

RESULTS OF INJECTIONS OF KILLED CULTURES. During the investigation, 37 patients were injected with streptococci killed by heat. The number of cocci used for injection varied from 25,000,000 to 100,000,000. The effect on the opsonic index was studied in 10 patients receiving 25,000,000 cocci, in 5 patients receiving 50,000,000 cocci, and in 11 patients receiving 100,000,000 cocci. The results are indicated in the following tables with the opsonic index, before and after injection.

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[1] Jour. Amer. Med. Assoc., 1906, xlvi, 108.

Opsonic Index of Patients Receiving 25,000,000 Streptococci.

	Before vac.	1 day after vac.	2	3	4	5	6	7	8	9	10
1	1.1	...	0.7	...	...	1.2	1.2	2.8			
2	1.0	3 0	2.1	1.7							
3	1.7	...	...	...	0.9	...	0.8				
4	0.6	...	...	1.5	...	0.7	4.1				
5	1.0	...	1.0	0.9	...	...	...	0.9		0.8	
6	0.7	...	1.3	...	0.9	1.1	...	1.0			
7	2.4	...	0.8	2.1	2.5	1.7					
8	1.2	...	1.3	1.0	...	1.1	1.7	1.1			
9	0.4	1.4	...	...	1.3	...	1.1	...	0.8		
10	0.3	1.1	...	1.7							
Aver.	1.04	1.83	1.2	1.48	1.4	1.16	1.78	1.45	0.8	0.8	

Opsonic Index of Patients Receiving 50,000,000 Cocci.

1	1.0	0.8	...	0.8	...	...	...	...	0.7		
2	1.4	1.3	...	...	...	...	...	...	...	1.0	
3	0.8	1.4	1.2	...	0.8	...	1.2				
4	1.1	1.0	1.2	...	1.2	...	1.1	...	0.9		
5	0.6	0.4	...	1.0	...	1.1	...	1.0	0.9	...	1.1
Aver.	0.98	0.98	1.2	0.9	1.0	1.1	1.15	1.0	0.83	1.0	1.1

Opsonic Index of Patients Receiving 100,000,000 Cocci.

1	1.2	...	...	0.5	...	0.5	...	1.3	0.5	0.6	0.8
2	1.0	1.0	...	0.9	...	0.8					
3	1.0	...	1.1	...	...	0.8	1.9				
4	1.1	0.3									
5	0.9	...	1.2	...	0.6	1.0	0.8				
6	1.0	0.8	1.3								
7	0.8	0.4	1.3	0.6	1 3	...	0.9				
8	0.8	...	1.2								
9	1.5	...	1.1	0.7							
10	0.6	...	1.0	1.0	0.9						
11	1.2	0 9	2.4	1.0	1.5	...	1.2	...	1.0		
Aver.	1.029	0.68	1.325	0.74	1.08	0.825	1.2	1.3	0.75	0.6	0.8

Some of the results of vaccination on the opsonic index are summarized in the following table:

	25,000,000.	50,000,000.	100,000,000.
Index rose without preceding fall	5	1	4
Index fell with subsequent rise	3	1	4
Index remained unchanged	1	2	1
Index fell and remained depressed	1	1	2

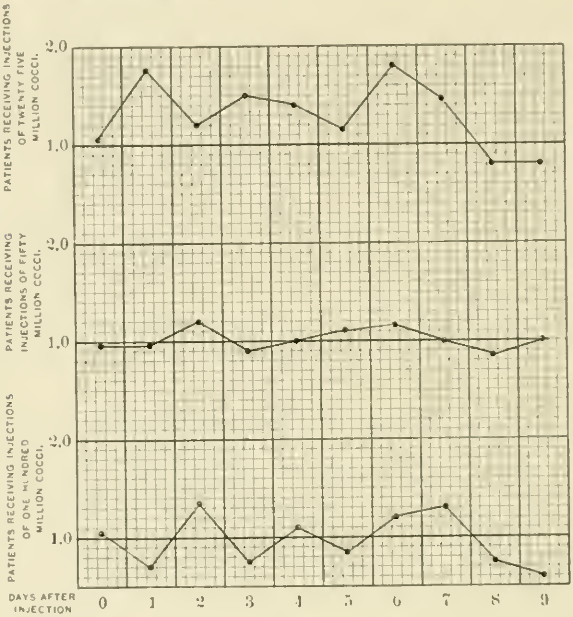

CHART IV. To show the average effect of vaccination on the opsonic index

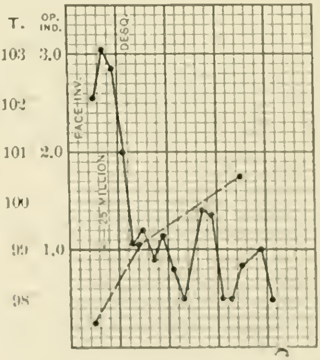

CHART V. H., an adult male, had been suffering with coryza for about two weeks previous to admission. He had a chill on the day of admission and developed erysipelas of the face. He received one injection of 25,000,000 cocci on the first day of the disease. (Temperature, unbroken line; opsonic index, broken line).

The effect of vaccination on the opsonic index is represented by Chart IV, which shows the average of the opsonic indices for the different days after vaccination with 25,000,000, 50,000,000, and

100,000,000 cocci. Charts V, VI, and VII are from individual cases and show rise of opsonic index after injection of varying amounts of killed streptococci.

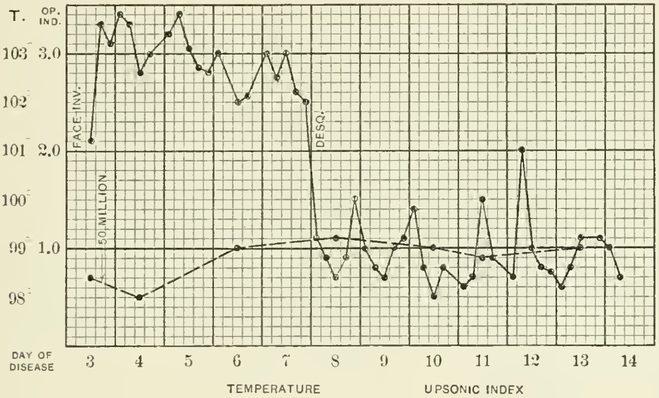

Chart VI.—C., an adult male, suffering with erysipelas of the face and delirium, received an injection of 50,000,000 cocci on the third day of the disease. (Temperature, unbroken line; opsonic index, broken line).

When 25,000,000 cocci are injected, not only is there no negative phase of the opsonic index, but the index is elevated after twenty-four hours. On the day after the first elevation, there is a fall, but the index maintains a level considerably above normal during seven

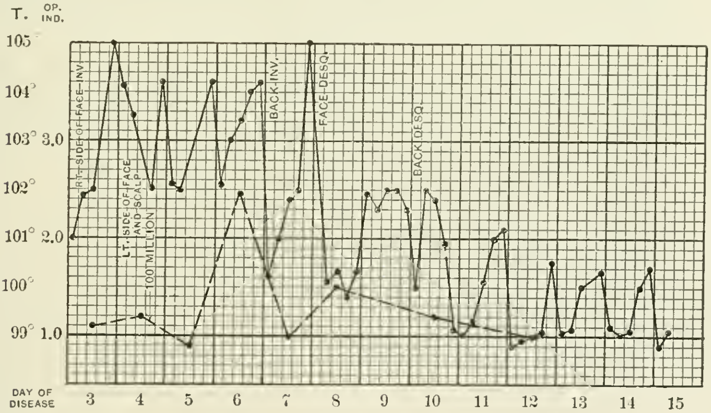

Chart VII.—M., a woman, twenty-three years old, was admitted on the third day of the disease with erysipelas of the right side of the face. Two days before admission she had had a severe chill, felt nauseated and weak. She received 100,000,000 killed cocci on the fourth day of the disease. (Temperature, unbroken line; opsonic index, broken line.

days. Only one-half of the patients discharged from the hospital as cured have a higher index on discharge than on admission.

The opsonic index determined with streptococci from patients with erysipelas shows no constant change at the time of desquamation. At the time of desquamation, the opsonic index was higher than it had previously been in 8 of 13 cases, and twice it was lower than it had been at any time during the disease.

It is further of interest that of 33 patients whose indices were determined, the indices of 22 of them fell after desquamation. Of these, 2 have been again admitted to the hospital with recurrence.

Of 3 cases of recurrent erysipelas, in 2 there was a lower index at the time of desquamation than there had been before desquamation. One patient had a higher index at the time of the first desquamation and a lower one at the time of the second desquamation than had been observed in the case before.

REPEATED INJECTIONS OF KILLED STREPTOCOCCI. Several cases of migratory erysipelas and several with complications, such as otitis media and pyemia, received more than one injection of killed cultures. In these cases the first injection produced no marked increase of the opsonic index. The second injection was made after an interval of from three to ten days, and was usually smaller than the first. In most cases, the index ultimately rose.

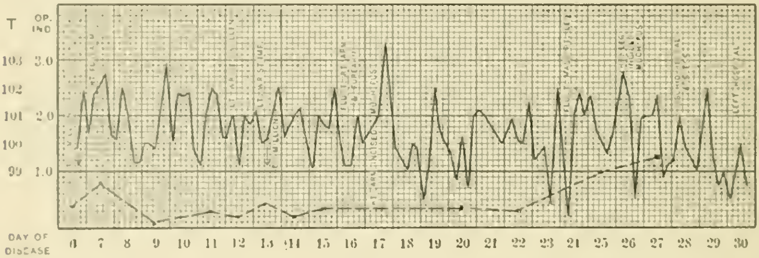

CHART VIII.—F., a woman, thirty-one years old. Five days before admission the patient's right arm began to swell and became red. She felt weak and feverish, and had numerous attacks of vomiting. There was no history of chill or trauma. This patient received an injection of 100,000,000 cocci on the sixth day of the disease, and 50,000,000 cocci on the thirteenth day of the disease. Complications were pyemia and apical tuberculosis. (Temperature, unbroken line; opsonic index, broken line).

In 4 injected patients whose opsonic index was determined at intervals, an intercurrent disease developed. Of these, 2 had pneumonia, 1 pyemia (Chart VIII), and 1 died with the diagnosis of tuberculosis and nephritis (Chart IX). In all of these patients the index rose with the development of the complication. The rise was especially marked with the appearance of pneumonia (Chart X). The significance of this cannot be determined, since only 1 patient with pneumonia died and no autopsy being made, the microorganism causing the pneumonia could not be determined.

SUMMARY OF CLINICAL RESULTS. *Temperature.* In the first few hours following an injection of killed cultures, there may be a trivial rise of temperature from 0.2° to 0.6° F. There can be no definite conclusions as to the permanent effect on temperature.

Delirium. A large number of the injected patients, as well as those not injected, were delirious at times, so that a conclusion regarding the relative frequency of delirium after injection is not possible.

Desquamation. Of 21 uncomplicated cases of erysipelas treated by the injection of killed cultures, injections were made twice on the first day of the disease; these patients desquamated on the second and seventh days, respectively. The average duration from onset to desquamation when treated by injections of antistreptococcic serum, as reported by Dr. Ayer,[2] in 1905, was seven and one-half days. Two cases of the present series were injected on the second

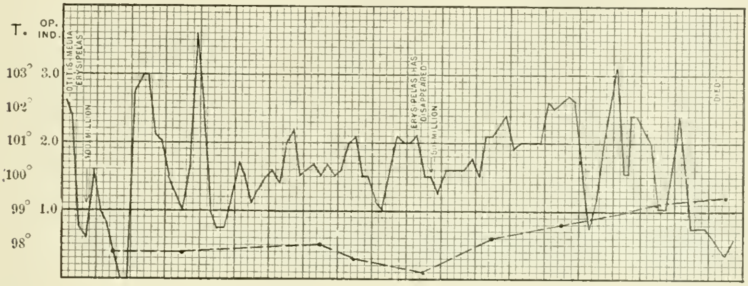

Chart IX.—H., a male, was admitted to the hospital December 3, 1906, with cystitis. On December 27 he developed otitis media, and on January 6 erysipelas of the face. Blood cultures were found to be negative. On January 10 he received 100,000,000 streptococci, again on January 16, 100,000,000, and on January 26, 50,000,000. He was never cured of his otitis media. He died February 5 with a diagnosis of apical tuberculosis and nephritis. (Temperature, unbroken line; opsonic index, broken line)

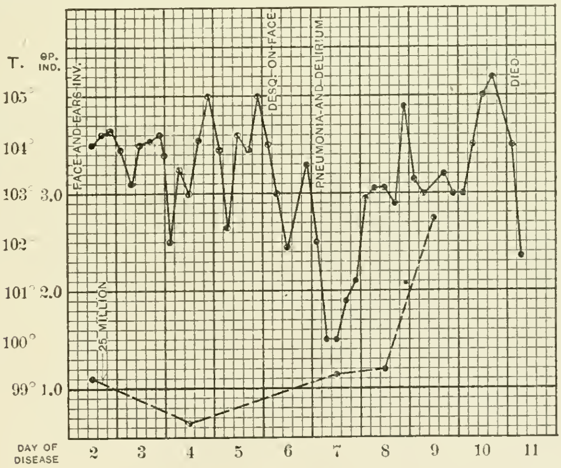

Chart X.—G., a man, thirty-four years old, was admitted on the second day of his disease. On the day before admission the patient had had a severe chill; his face began to burn and became red. He received 25,000,000 killed cocci on the day of admission; on the seventh day the disease was complicated by pneumonia. The patient died on the eleventh day. No autopsy was obtained. (Temperature, unbroken line; opsonic index, broken line).

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[2] Medical Record, 1905.

day of the disease and desquamated on the fourth and sixth days, respectively; an average of 10 cases injected with antistreptococcic serum on the second day by Dr. Ayer desquamated on the seventh day. Four cases injected with killed cultures on the third day of the disease desquamated on the fifth, fourth, fifth, and eighth days, respectively; an average of five and one-half days as compared to an average of six and nine-tenths days, as observed by Dr. Ayer in 21 cases receiving antistreptococcic serum on the third day. Six patients were injected on the fifth day and desquamated on the fifth, eighth, fifth, eighth, eighth, and tenth days of the disease. Two patients were injected on the fifth day of the disease and both desquamated on the seventh day. One patient was injected on the sixth day and desquamated on the seventh day. Three patients were injected on the seventh day and desquamated on the eighth, ninth, and tenth days, respectively. One patient injected on the ninth day of the disease desquamated on the eleventh.

The average duration of the disease in the 21 cases in this series is six and eight-tenth days. In 79 cases of erysipelas which received only local treatment, Dr. Ayer found the average duration of the disease to be nine and four-tenth days, which is two and eight-tenths longer than that of the present series. Of 48 patients receiving antistreptococcic serum, the average duration was found to be about the same as in this series.

The relation of dosage of streptococci to desquamation was as follows: Those receiving 25,000,000 cocci desquamated three days after injection; those receiving 50,000,000 cocci desquamated three and one-half days after injection; and those receiving 100,000,000 cocci desquamated two and four-tenths days after injection.

Migration. Migratory forms were found in 8 patients who had received injections of killed streptococci. Whether this migratory form was caused by injections of cultures cannot be determined, since other cases of the migratory type were observed when no injections were made. One of the injected patients with this form of the disease died; this patient had previously received 60 c.c. of of antistreptococcic serum and received one injection of killed cultures of streptococci three days before death. The disease, however, was complicated with chronic interstitial nephritis.

It is quite clear that injections of killed cultures of streptococci from erysipelatous lesions do not prevent migration, even if the opsonic index rises.

Recurrence. Recurrence was observed in a number of cases treated with injections of killed cultures. Since February 15 two of the patients included in this investigation have been again admitted because of recurrence. It is well known that an attack of erysipelas will not prevent a second attack of the disease, hence it is hardly to be expected that the injections of killed cultutes will prevent future attacks. Of the 37 patients who received injections of killed cultures, 3 died. In all of these the erysipelas was complicated by some other disease.

The opsonic index is subject to such great irregularity that its determination gives little indication of the severity of the disease and is of no value for prognosis. There is in most cases of erysipelas an increase of the opsonic index occurring most often during the first three days, and this rise is followed by a fall. The injection of killed streptococci causes, on the whole, an increase of the opsonic index, but a relation between the elevation of the index and the improvement of the patient was not observable.

It is difficult to determine the value of the injection of killed cultures of streptococci in erysipelas. While this injection does not prevent migration and recurrence, the apparent shortening of the duration of the disease suggests that injections have some value. Yet the effect upon the duration of the disease is uncertain because during the time of our investigation no suitable cases remained untreated with which comparison could be made, and it is known that the duration of the disease varies in different years.

This investigation was made possible through the kindness of Dr. J. C. Ayer. I wish to express my obligation to Dr. Ayer and to Dr. Gordon Lindsay, the house officer in Bellevue Hospital.

REVIEWS.

Physical Diagnosis, with Case Examples of the Inductive Method. By Howard S. Anders, A.M., M.D., Assistant Professor of Physical Diagnosis in the Medico-Chirurgical College, Philadelphia. Pp. 456; 88 illustrations in the text, and 32 plates. New York and London: D. Appleton & Co., 1907.

Dr. Anders desires to improve the student both in his technique and in his reasoning processes—two things in which, as he justly contends in the preface, the ordinary student is lamentably weak. He holds that the work of the diagnostician is akin to that of a detective, and he has attempted, therefore, to apply the principles of inductive reasoning to physical diagnosis. He limits physical diagnosis to those diseases of the chest and abdomen which can be detected "by the aid of signs denoting the abnormal physical condition of the organs or structures under examination." This excludes all those objective phenomena occurring in diseases of the nervous system, of the joints, muscles, etc., which, of course, should be regarded as physical signs.

As a whole, the book is very good. The style is clear, the arrangement logical, the illustrations fairly demonstrative. The majority of the latter are borrowed, although there are 11 original plates in the body of the book somewhat similar to those in Musser's work, and 21 plates showing a considerable number of Röntgengrams of the chest and abdomen, taken by Dr. Pfahler, which are excellent.

After a section devoted to topographical anatomy, Dr. Anders takes up inspection; but he fails to note that a normal thorax may co-exist with congenital anomalies of the lung and marked displacement of the heart. It is not clear why he says that rapidity of breathing is "not strictly a physical sign in itself." He recognizes pulsating empyema only on the left side, although it has been observed on the right. In the section on palpation he lays great stress upon the value of fremitus as a physical sign. He includes under this not merely the vibrations produced in the chest wall by the spoken voice, but all other vibrations, whether due to rales or friction, which may be felt by the hand applied to the body. The section on auscultatory percussion might have been somewhat fuller, and some of the factors causing inaccuracy should have been mentioned. It might be inferred that percussion over the ribs should be avoided.

The most unsatisfactory part of Dr. Ander's book is his discussion of the subject of pitch. This is partly to be explained by the fact that of all modifications of physical signs it is the one responsible for the greatest amount of confusion and difference of opinion. In the present work the confusion is by no means made clear. For instance, we have the sweeping statement that "pitch is the most important element in the differential diagnosis of air-less from air-containing tissues." Then, "the transition from clearness to dulness is realized more by the gradual elevation of pitch, than by any other acoustic attribute." On the next page we find "the louder or clearer the sound, the lower the pitch. The higher or shriller the sound, the less its loudness." But the student is cautioned "not to confuse the high-pitched, dull sound with the high-pitched tympanitic note." There is then a long discussion of the pitch of the percussion note over the lungs in normal and in morbid conditions, and particularly the modifying effect of tension, which does not serve to make the subject clearer; and in no place does Dr. Anders recognize what is rather the more common opinion of the present day, that pitch and loudness or intensity are essentially different factors, although, under certain circumstances, they may vary together. The eagerness to use pitch as an important element in physical diagnosis becomes almost absurd when it is applied to such a complex noise as the succession splash (p. 186), and its variation used for differentiation.

There is a seeming misstatement on page 13, that in retraction of the lungs, due to a pushed-up diaphragm, the borders of the lungs are motionless. The motion of the lower borders, under these circumstances, is one of the most trustworthy signs of differentiation from pneumothorax. Dr. Anders lays great stress upon auscultation directly, as well as with the stethoscope. He does not pay enough attention to the different kinds of information furnished by the different types of stethoscope, nor does he explain in sufficient detail the types of sound heard best with the ear, and those heard best by mediate auscultation. He believes that the transmission of the whispered voice is often more valuable than the transmission of the spoken voice. He does not differentiate very clearly between the different types of pectoriloquy, although he lays a good deal of stress upon the variations in the pitch.

In his discussion of physical diagnosis of the heart, it is necessary to take sharp issue with the statement that "when the alteration of the form of the precordium is more general, the prominence more marked, and unmistakable fulness of the intercostal spaces evident, it points to the presence of a pericardial effusion." Actual bulging of the precordium, involving the bony structures, as a result of pericardial effusion is extremely rare. Again, systolic retraction in the neighborhood of the nipple cannot be dismissed with the statement, "its significance is adhesive pericarditis," for it occurs in other conditions. Hypoplasia of the lung is not mentioned as a cause of cardiac displacement.

Dr. Anders follows the prevailing view in preferring the deep to the superficial cardiac dulness. It is rather interesting to read the direction that, during the examination of the heart, "the patient should be at rest bodily, and serene in mind." On page 276 stress is laid upon the pitch of the second sound in contradistinction to its intensity. There is a very good, although brief, discussion of cardiac rhythm.

In the discussion of the various points relating to the physical signs of heart disease, it is necessary to begin by disputing vigorously the statement (page 287): "Given these two physical signs [area of greatest intensity and time of the murmur] in conjunction with the anatomical and physiological data related thereto, and the pathological significance is virtually a matter of logical deduction with syllogistic regularity." This may be compared with the more conservative statement of Fraentzel, who, in his later life, felt that after four or five hours devoted to the careful study of a case of heart disease, he could sometimes feel reasonably sure of the diagnosis.

In discussing the murmur of mitral stenosis no mention is made of the interrupted diastolic murmur, perhaps the most characteristic of all the varieties. The systolic murmur heard in the left axilla in mitral regurgitation is ascribed simply to the reflux of blood through the pulmonary veins. In aortic regurgitation no mention is made (excepting in one of the hypothetical cases on page 360) that in nearly all cases a systolic, as well as a diastolic, murmur is heard—a fact recognized by all the earlier writers, and to which Tyson, among the modern authorities, gives emphasis. In discussing tricuspid insufficiency no mention is made of pulsation of the liver as an important physical sign, though this is twice mentioned in connection with diseases of the liver. Among the rarer murmurs should have been mentioned the systolic vascular murmur that is sometimes heard in brain tumors, and in organic disease of the meninges and the skull.

The final section of the book is devoted to the examinations of the organs of the abdomen. Dr. Anders makes the original suggestion—at least it is new to us—of dividing the abdomen into six regions by lines radiating from the umbilicus. This seems, in all respects, a logical and practical method, and the best that has hitherto been suggested. Each of the six regions is in a sense an entity, containing organs that, clinically, are properly grouped together. The rest of this section, however, cannot be regarded as satisfactory. It is rather a catalogue of the various conditions that may occur, than a precise description of the physical signs which lead to their recognition. The ovarian cyst is not mentioned as one of the difficulties in the diagnosis of ascites. The interesting physical signs of subdiaphragmatic abscess and pyopneumothorax are not even mentioned. The value of the study of the pulsation of the right common iliac artery in appendicitis is ignored. Retroperitoneal sarcoma is said to project in the right lower sextant.

In the diagnosis of the position of the stomach, numerous methods are altogether omitted, including auscultatory inflation introduced by Musser. In a discussion of the auscultation of the stomach and intestines the revolutionary work of Cannon has not been mentioned, and auscultation, a most valuable method, is dismissed with a brief paragraph commencing with the statement that it is of little diagnostic value. The value of the absence of liver dulness as an indication of pneumoperitoneum, is forgotten. The differential diagnosis of the different types of renal tumor is dismissed with a brief paragraph. The physical examination of the intestines occupies two and one-half pages, and is wholly inadequate.

A very successful effort has been made to include accurate descriptions of the important physical signs to which proper names have been applied. This, with an excellent index, makes the work valuable for reference. J. S.

A Text-book of the Practice of Medicine. By Hobart Amory Hare, M.D., B.Sc., Professor of Therapeutics in the Jefferson Medical College, Philadelphia. Second edition; pp. 1132; 131 engravings and 11 plates in colors and monochrome. Philadelphia and New York: Lea Brothers & Co., 1907.

The fact that reprintings and a second edition of this book have been so quickly called for is evidence of its popularity. The general character of the work is sufficiently well known from the previous edition. This new edition has evidently been extensively revised, in conformity with the movement of medical investigation; and in the great majority of instances the most recent work is included and referred to: for example, the status of Treponœma pallidum in relation to syphilis, the relation of the Leishman bodies to kala-azar, and many other recent investigations, are in the text, and have their proper weight given them. In questions concerning treatment, the author naturally has distinct and often very independent ideas; and in many particulars his observations regarding treatment are extremely sensible and valuable. For example, many persons still need seriously to consider carefully the importance of observations such as those made concerning the treatment of chronic nephritis, and especially the dietetic treatment of this disease; though it would have been well, from the reviewer's point of view, if, in this instance, some attention had been given to the increasing belief that high arterial tension usually has a purpose, and, while nature may overdo this purpose, it probably is not best for the therapeutist to go so far as to keep the tension "at a point as near the normal level as possible." This however, is, still a question, to some extent, of one's point of view. The book in general is another illustration of the author's well-known capacity for collecting a vast number of facts and compressing them under one cover into an interesting systematic treatise. D. L. E.

SURGICAL DIAGNOSIS. By DANIEL N. EISENDRATH, A.B., M.D., Adjunct Professor of Surgery in the Medical Department of the University of Illinois, Chicago, Illinois. Pp. 776, with 482 illustrations. Philadelphia and London: W. B. Saunders Co., 1907.

THAT diagnosis is of vast importance no one can deny; but it is, after all, only the first step toward treatment. For as the silver hid in the mines of greedy Earth is colorless until minted and put to proper use, and as the cooking of food is futile unless it be eaten, so for a surgeon to be able merely to diagnosticate a surgical affection is for him to fall short of his highest functions. Any monograph, therefore, which deals with diagnosis alone, leaves the reader with aspirations unfulfilled, with desires unappeased, and with numerous riddles unsolved. Not only do writers on diagnosis thus lop off, as it were, their most useful hand, but those who adopt the plan of Dr. Eisendrath abstract as well the mind whose ratiocinations give the impulse by which the hand is moved. Dr. Eisendrath, by confining himself rigidly to the symptomatology of injury and disease, excludes from consideration, practically in their entirety, the subjects of etiology and pathology, without which the science of diagnosis becomes a mere art, with a dangerous tendency toward those snap diagnoses which are so brilliant when correct, and so humiliating when wrong.

Even with these disadvantages, inherent in the plan he has adopted, Dr. Eisendrath has written a notable book, and one for which the profession owes him a debt of gratitude. The illustrations form what publishers are wont to call a "feature" of the volume, and are in our opinion its most valuable portion. Indeed, we deem it a pity that the wealth of illustration is not even more profuse, and that the text has not intentionally been made to assume a subordinate role. The work would thus have become more of an atlas than a text-book, and had the author inserted the clinical histories of the patients represented, instead of attempting a systematic treatise on symptomatology, we doubt not that the usefulness of the volume would have been considerably augmented. In the limited space at his disposal—diminished very appreciably by the elaborate system of paragraphing and display-typing which is employed—Dr. Eisendrath has been forced to treat many subjects in so compendious a manner that both the earnest student and the careless crammer will be forced to search elsewhere to satisfy himself that he has mastered the affection in question. An example is that although it is succinctly stated that injuries of the phrenic nerve cause paralysis of the corresponding half of the diaphragm, yet nowhere is there to be found a description of the symptoms present in diaphragmatic palsy. The mere fact that this is produced by lesions of the phrenic might be assumed to be familiar to a student who had acquired even a modicum of antomy, but it is far less easy to infer what symptoms will be produced. Those sections which have impressed the reviewer as least open to this criticism are those on the female breast, and on acute abdominal affections. The portions of the work treating of the injuries of the extremities are also excellent, especially praiseworthy being the illustrations showing the methods of examination in the diagnosis of fractures. Dr. Eisendrath, however, has fallen into a curious error in connection with elbow fractures: he speaks of Fig. 304, p. 472, as representing a fracture through the epiphysis, and refers to it in the text as an epiphyseal separation, whereas the illustration very clearly represents the familiar type of fracture known as the transverse diacondylar, and the structure called in the legend "upper epiphysis (capitellum) of radius" is no other than the epiphyseal centre for the capitellum of the humerus. Between this epiphyseal centre and the shaft of the humerus lies the epiphyseal line, and an epiphyseal separation occurs along this line. This error appears the less excusable, as the author frequently in the text cautions against errors in diagnosis from skiagrams, due to ignorance of the true position of the epiphyseal lines. Many of the skiagrams in the volume have been so touched up with outlines, etc., that their value as skiagrams is destroyed, while their usefulness is not as great as would be the case were they replaced by real diagrams.

It is to be hoped that in another edition, which will doubtless soon be demanded, the author will take pains to correct numerous grammatical as well as rhetorical errors in the text. The newspaper English employed is not only unscholarly, but it is ambiguous, *e. g.*, "They (fibromas) occur almost exclusively in the lower jaw, and until there is a prominence over the bone the patient is not aware of its presence." It is as a rule only extremely intelligent patients who can be expected to be aware of the presence of that which does not yet exist. The author speaks of "sequestræ," of distinguishing "a periosteal fibromata from a periosteal sarcomata," and finally informs the reader of page 535 that "if death ensues from the immediate virulence of the infection, the case may pursue a more protracted, typhoid-like course." A. P. C. A.

A Text-book upon the Pathogenic Bacteria. By Joseph McFarland, M.D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College, Philadelphia. Fifth edition; pp. 647; 190 illustrations. Philadelphia and London: W. B. Saunders Co., 1906.

Dr. McFarland's book upon the *Pathogenic Bacteria* has become so well and favorably known that there is little need of aught else than to chronicle the issuance of a new edition. The original plan of limiting the book to a discussion of the biological characteristics of the pathogenic bacteria has been adhered to, but much new matter has been added throughout the book, and that which had become obsolete has been eliminated. The chapters upon infection and immunity, of which our knowledge is rapidly increasing, have been entirely rewritten; they reflect very satisfactorily our present knowledge of the subjects. The book assuredly will continue to enjoy, as it merits, the favor of students and practitioners of medicine. A. K.

Progressive Medicine. A Quarterty Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, M.D., Professor of Therapeutics and Materia Medica in the Jefferson Medical College of Philadelphia, assisted by H. R. M. Landis, M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College, Philadelphia. Pp. 290, Vol. III, September, 1907. Philadelphia and New York: Lea Brothers & Co., 1907.

The first article in Vol. III, for 1907, of Progressive Medicine, is by William Ewart, who devotes 92 pages to an exceedingly interesting account of recent progress in diseases of the thorax and its viscera, including the heart, lungs, and bloodvessels. Among the especially important contributions are several on tuberculosis, including the report of the Royal Commission on human and bovine tuberculosis, the intestinal and glandular origin of pulmonary tuberculosis, the treatment of tuberculosis, etc.; the high diaphragm and its diagnostic significance in regard to the blood and the circulation; the diagnosis and treatment of pleural effusions; bronchoscopy as an aid to diagnosis in pulmonary diseases; the physical signs of diseases of the lungs, pleura, and heart; the diagnosis of disorders of the myocardium; disorders of the pulse; cardiac arrhythmias and heart-block; and the treatment of diseases of the heart. William S. Gottheil devotes 39 pages to recent progress in dermatology and syphilis, beginning with an important series of dermatological axioms, and continuing with a discussion of carcinoma and endothelioma of the skin, feigned eruptions, the diagnostic and prognostic value of herpes, disorders of the nails, and syphilis in its protean manifestations. Edward P. Davis devotes 105 pages to a review of recent progress in obstetrics, comprising especially ectopic pregnancy, the toxemia of pregnancy, the complications of pregnancy, placenta prævia, septic infection, and abortion; labor, normal and abnormal; obstetrical surgery, etc. William G. Spiller devotes 40 pages to recent progress in diseases of the nervous system, especially brain tumors, aphasia, cerebral hemorrhage, tabes, paresis, multiple sclerosis, tumors of the spinal cord, epilepsy, etc. On the whole, the volume is exceedingly valuable and interesting, a worthy member of a series that should find a place on the table of every practising physician. A. K.

A MANUAL OF THE DIAGNOSIS AND TREATMENT OF THE DISEASES OF THE EYE. By EDWARD JACKSON, A.M., M.D., Professor of Ophthalmology in the University of Colorado. Second edition; pp. 615; 182 illustrations and 2 colored plates. Philadelphia and London: W. B. Saunders Co., 1907.

THIS the second edition of Jackson's *Manual* has been brought thoroughly up to date. The student who masters it will have little that books can teach him to learn of the diagnosis and treatment of the diseases of the eye. The subject is presented with the clearness and sense of proportion to be expected from a teacher and writer like the author.

About one-sixth of the work deals with optics as applied in the study of the physiology, diagnosis, treatment, etc., of the eye—the portion which especially differentiates the study of the organ of vision from the other organs of the body. And this part of the subject is dealt with by a master. The refraction of light is made clearer, as the phenemena are referred to the waves as well as to the rays. The author truly remarks that the examination with the trial lenses requires care, system, experience, and good judgment; some excellent rules are given for the attainment of accurate results. We could wish that Dr. Jackson had been somewhat fuller in his account of the astigmatic lens—this capital device employed by him, for accurate and rapid subjective examination.

The somewhat picturesque suggestion is thrown out that homatropine or euphthalmine may be of service in some cases in making a diagnosis of incipient glaucoma (by causing an outbreak at a selected time favorable for operation). Cocaine is stated to enhance the effect of myotics in lowering intra-ocular tension. The removal of the clear lens in high myopia is still recommended in special cases, that is, when after trial it is found that the correction by glasses cannot be borne.

Judicious views characterize the work throughout. The extravagances which have been exploited in practical ophthalmology from time to time find no countenance. In short, this manual is an excellent exposition of the present state of ophthalmic science, and is to be warmly recommended to every one interested in the subject.

T. B. S.

THE PRACTITIONER'S MEDICAL DICTIONARY. By GEORGE M. GOULD, A.M., M.D., author of an *Illustrated Dictionary of Medicine, Biology, and Allied Sciences*; editor of *American Medicine*, etc. Pp. 1043; 388 illustrations. Philadelphia: P. Blakiston's Son & Co., 1907.

THE *Practitioner's Dictionary* is announced as being new in every respect and detail—not a revision of the other dictionaries that have made Dr. Gould's name well known throughout the medical world. The avowed object of the new book is to supply the practitioner with trustworthy definitions of such medical words and terms as he may need in his reading of standard medical text-books, monographs, and journalistic literature. The old and accepted features of previous lexicons have been retained; of these, the chief is a simple method, easily understood at a glance, of indicating the pronunciation and accentuation of words; and a second is the tabulation and classification of correlated terms. Of the last mentioned, however, there is one notable exception—the distribution in alphabetical order throughout the book of eponymic terms, believed to be preferable to tabulation on account of the great increase in these terms. Several valuable and important novelties have been introduced in order to make the book meet all the latest requirements: The metric system of weights and measurements in doses, etc., has been added in order to furnish the necessary equivalents to those who may prefer these to the older and more common forms; the additions and changes to the new Pharmacopœia have been inserted; and the new Basle anatomical nomenclature has been incorporated. Of the book, as a whole, one can only say that it is excellent, that it comes up to one's expectations, and fully meets all requirements. It is the most compact book of its kind—printed of large, clear type, on thin, tough, opaque paper, so that it is not more than an inch and a half thick. Its many excellent features doubtless will secure for it quite as great a measure of success as has been attained by the other dictionaries prepared by its distinguished author

A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF
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The Phagocytosis of Erythrocytes and the Question of Opsonins in Paroxysmal Hemoglobinuria.—J. Eason (*Edin. Med. Jour.*, 1907, xxi, 440) states that most of the important work under discussion at present is on the determination of the identity of the substance in the blood which exercises the opsonic function and whether this differs from the amboceptor or immune body, or is like it. Barrett gave the name hemopsonin to that substance causing phagocytosis of erythrocytes. In paroxysmal hemoglobinuria hemolysis depends on the activity of an intermediary body or amboceptor whose activity is in abeyance if the blood is maintained (in vitro) at normal temperature. Under such circumstances no hemolysis cocurs, but if such blood is cooled after being mixed with potassium oxalate to prevent coagulation, and then maintained at 37° C. in an incubator for three hours hemolysis follows. Control tubes of normal blood under similar conditions show no hemolysis. Active solution of the erythrocytes by phagocytes can be seen in the pathological blood after thirty minutes in ice-water and a similar period at 37° C. This process may be partly extracellular, as by contact with the leukocytes the erythrocyte is dissolved and the hemoglobin absorbed. "Phagolysis sometimes appears to be proceeding as rapidly as hemolysis, and the erythrocytes appear to undergo very rapid solution in the medium (complement?) thus set free from the leukocytes. This process may also be observed if the inactivated serum of a case of paroxysmal hemoglobinuria is mixed with washed corpuscles and leukocytes from a normal individual and is submitted to the conditions of temperature already described. This form of phagocytosis would, therefore, not appear to be dependent on the action of a thermolabile substance or normal opsonin, but on a thermostabile substance which may be either the hemolytic amboceptor or a body corresponding to the thermostabile opsonizing substance which may be artificially produced by immunizing an animal."

These observations may be made clinically by employing the method originally used by Ehrlich to simulate the condition of a paroxysm as follows: A ligature is put around the finger and then the finger put in ice-water (5 to 10 minutes) and then in warm water a similar length of time. Blood smears are then examined. Eason concludes as follows: "Thus it appears that phagocytosis is as much dependent on the previous chilling of the blood as is the extracellular lysis; another indication that the amboceptor is the cause of both processes."

On the Behavior of the Eosinophile Leukocytes in Cases of Pulmonary Tuberculosis.—JOHN M. SWAN and HOWARD T. KARSNER (*New York Med. Jour.*, 1907, lxxxv, 539). One of the authors (Swan) in 1904, in a study of the blood in 25 cases of pulmonary tuberculosis, came to the following conclusions: "The absence of the eosinophiles from the blood may be looked upon as an unfavorable prognostic sign. The increase of these cells while the patient is under treatment may be taken as an indication that the progress of the disease has a tendency to become arrested." This present study has to bear especially on this point and consists of the differential counts in 31 cases of pulmonary tuberculosis. In all cases 500 leukocytes were counted and the specimens were stained by Wright's method. The studies are from the Philadelphia General Hospital. In bedridden patients counts were made once a week or oftener if the case was ending fatally. In those not in bed, monthly counts were made. In a series of 31 cases, 26 were fatal and 5 were stationary or improved at the time the observations were discontinued. With the exception of 2 cases, the eosinphile cells were below 1 per cent. as a rule at periods varying from 102 days before death to the day of death. In these cases they varied from 1 to 2 per cent. for more or less lengthy periods before death. In the typically fatal cases the count was usually under 1 per cent.—as low as 0.2 per cent.; and the cells were even absent just before death. In the stationary or improved cases the eosinophilic count usually rose until discharge or discontinuance of the observations. The authors show interesting tables and charts and then append the counts of all cases in detail. They conclude as follows:

"In the light of these studies we should say that in cases of pulmonary tuberculosis the eosinophile cells tend to disappear from the circulating blood as the progress of the disease brings the fatal termination nearer, and that as the patient improves under treatment and as the disease shows a tendency to become arrested, the eosinophiles reappear in the circulating blood. It appears that this tendency of the eosinophile cells up and down may be of some use as a prognostic sign. It appears that this fluctuation of the eosinophile cells is a measure of the severity of the secondary infection with pyogenic organisms and not an indication of the extent of the tuberculous process itself; in fact the reappearance of eosinophilic cells in the circulating blood coincident with the improvement in the cases of phthisis is probably due to the removal of the chemotactic influence of the products of growth of the pus organisms and the reassertion of the influence of the tuberculin manufactured in the tuberculous lesions."

Pre-icteric Itching.—D. Riesman (*Amer. Med.*, 1907, ii, 79) gives a brief *resume* of the literature bearing on this interesting subject and notes that, in many reported instances, the itching preceded the jaundice by some time. He then reports in detail three cases of jaundice due to different causes, in all of which pre-icteric itching was marked. Riesman thinks that the older view that itching is due to a deposit of bile pigment in the skin can no longer be maintained. This has also been the view of several other writers. Rolleston also doubtes whether itching is due entirely to the presence of bilirubin and admits the possibility that other concomitant poisons may give rise to it or that the dry, ill nourished condition of the skin is the cause. The following facts seem to bear out the view: (1) Itching according to Frerichs and Lancereaux occurs in only about one-fifth of the cases of jaundice. (2) It bears no constant relation to the depth of the jaundice. (3) It may come and go repeatedly in a given case, while the jaundice persists undiminished. (4) The occurrence of itching that appears before the jaundice and coincidently runs with it for some time. (5) The prompt, sometimes instantaneous disappearance of the itching after drainage of the gall-bladder long before the jaundice has had time to disappear.

The cause of the itching is unknown. In all probability it is some metabolic poison or poisons which the liver in its normal state either neutralizes as it passes through in the portal blood or destroys when brought to it by the blood of the hepatic artery. The poison must be the bile, as shown by the sudden ceasing as soon as the bile is given free drainage either externally or internally. In ordinary icteric itching it most probably enters the circulation with the bile, probably by way of the lymphatics, but in pre-icteric itching or in itching depending upon hepatic disturbances without jaundice it passes into the blood independently. Riesman concludes as follows: (1) Itching sometimes precedes the onset of jaundice by a variable length of time. (2) This itching may cease the moment the jaundice is established or it may continue indefinitely. (3) Itching, whether pre-icteric or icteric, is not due to a deposit of bile pigment in the skin. (4) When itching exists for which no cause can be found, the liver, as a possible factor, should not be overlooked. (5) Pre-icteric itching is suggestive, though not pathognomonic of the existence of malignant disease involving the liver or the biliary passages. This last statement is borne out by the views of some eminent authorities, though the cases are too few for any positive opinion on this point.

The Treatment of Acute Failure of the Heart. — Charles Bolton (*Lancet*, 1907, i, 870) notes that acute failure of the heart may be brought on in two distinct classes of cases: (1) The failure may be due to some organic condition affecting the heart, the blood, the bloodvessels and, therefore, initiated in a purely mechanical manner. (2) It may be the result of some nervous disturbance, such as occurs in sudden death from vagus inhibition or in cases of tachycardia. Bolton, on account of time and space, deals mainly with the first class of cases on account of its greater frequency and importance. Acute failure of the heart is brought about in two ways: By any condition interfering (1) with the emptying of its cavities during systole or (2) the filling of its cavities during diastole. As regards the first of these conditions the loss of muscular power may be due to many acute infectious diseases or other febrile conditions, or to direct disease of the musculature of the heart, either acute or chronic. Also among the influences bearing on this first condition are the various arterial or visceral changes leading to increased resistance to the output. The second condition, interference with the diastolic filling of the heart, may be brought about by an increase or diminution in the quantity of the blood, such as may occur in various conditions.

In regard to the treatment of the acute degenerative changes Bolton lays down three indications: (1) To prevent, to stop or diminish the degenerative process; (2) to diminish the work of the degenerate heart; (3) to simulate the heart only over very short periods when fatal syncope is threatened. Bolton lays great stress on the dangers of overstimulation, goes carefully into the general treatment of the different diseases, insisting especially on the avoidance of sudden movements and overstrainings and on the cautious treatment of such symptoms as bring about vomiting, as well as the relief of excitement and shock, and other means of avoiding sudden straining efforts. The importance of absolute rest is strongly emphasized. He then passes to the treatment of the condition causing acute failure of the heart by an increase in the diastolic quantity, such as is found after undue exertion and overstraining, and the effects of altitude upon the heart, and lastly takes up the conditions causing diminution of the diastolic quantity either by vasomotor paralysis, severe hemorrhage, or increased pressure in the chest acting on the heart. This latter is seen in pericardial and pleural effusions. Shock as one of the important factors in these conditions is treated of at some length. The general treatment, management, and drugs used in the various conditions for the relief of the many different symptoms reacting badly on the heart are considered at length.

The Spleen in Organic Heart Disease.—Ernest Barie (*La presse médicale*, 1907, xix, 145) calls attention to the fact that most of the organs have received much attention and study as to their changes in heart disease, in both compensated and uncompensated cases, but the study of the spleen has been much neglected. In his list of 216 personal observations of the conditions of the spleen in organic heart disease, 79 were carefully examined after death. The remainder were examined by careful measurements and other clinical methods before death which the author enumerates at length. In about two-thirds of the cases the spleen was of normal size, but in about one-third there were more or less changes, hypertrophy, diminution in size, the presence of infarcts. In the 20 cases of hypertrophy of the spleen the liver was usually enlarged in like extent. The spleen in 2 cases weighed 630 and 860 grams. The others weighed less and showed marked chronic passive congestion. In 4 cases the volume of the spleen was diminished, a marked atrophic condition being evident. Infarcts were found in 13 of the 79 autopsies. As a rule, in all the cases of abnormal spleen there were more or less serious diseases in the past history of the patient, and it is possible that they may have had some influence on the subsequent condition during a cardiac breakdown.

As regards the clinical signs and symptoms, those associated with the atrophic spleens are somewhat vague, while the diagnosis of infarcts was difficult; sudden local pain and slight enlargement of the spleen helped in their detection. In the cases of enlarged spleen a real "splenohepatic asystole" was induced and is characterized, contrary to the usual form, by a regularity of the pulse, absence of venous stasis, cyanosis, or oliguria, while, instead of the classical red and hard œdema, the œdema is white and soft. There was also pallor and frequent epistaxis, the latter sometimes being so severe as to require tamponing. Barié says: "Besides the pallor and the severe epistaxis, the progressive debility is the third chief important symptom in this 'splenohepatic asystole.'"

SURGERY.

UNDER THE CHARGE OF

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Internal Intestinal Fistulæ.—Hofmeister (*Beit. zur klin. Chir.*, 1907, liv, 745) says that abnormal communications between two small intestines or between a small and a large intestine are rare, aside from those produced by operative anastomoses. They have been found, chiefly, postmortem. In life they have been recognized in rare cases, as when a relatively free communication existed between an upper and a lower intestine. This might give symptoms that would render a diagnosis possible. Usually, however, the symptoms produced are not sufficiently definite to aid in the diagnosis. Among the causes of these internal fistulæ, according to Hilgreiner, are: tuberculous peritonitis, abscesses around a foreign body, actinomycotic abscesses, the breaking through of the closed end of a Meckel diverticulum. In the fistulæ between the small and large intestines carcinoma plays an important part. The peptic jejunal ulcer following gastro-enterostomy is of interest especially with regard to the possibilities of diagnosis and treatment. Hofmeister reports a typical case that has not hitherto been reported, in which the intestinal fistula had a traumatic origin. A boy, aged six years, suffered from many attacks of severe, paroxysmal, abdominal pain, difficult defecation, and frequent passages of blood in the form of lumps or masses. He became so emaciated, apparently from a deep-stated ulcerating stenosis of the intestine resistant to internal treatment, that one had to choose between leaving him to his fate or risking a fatal termination by operation, with the object of putting out of function the diseased portion of the intestine. This was done, but it did not help materially in the diagnosis, *i. e.*, of establishing the presence of a communication between the portion above and that below the artificial anus.

The mother had said that from time to time, since the artificial anus had been present, a movement by the rectum had occurred, but this fact had been overlooked. A second operation was done and a mass of adherent intestine was resected. This showed a fistula in that portion of the intestine leading to the artificial anus. An ileocolostomy was then performed, which was followed by undisturbed healing. The kick which the patient had received in the abdomen had evidently produced a wound of the intestine which led to a localized peritonitis with abscess formation between the neighboring intestines and perforation of the abscess on three sides. Healing of the abscess followed, but there was persistence of the fistula opening into three portions of the intestine.

The Technique of Pancreatectomy.—DESJARDINS (*Revue de chir.*, 1907, xxvii, 945) says that the pancreas for surgeons is a noli tangere, since they do not dare to remove it as they do other organs. It should not be regarded as so inaccessible as formerly, with the advantage of Mayo-Robson's cushion and his or Kehr's incision. The control of hemorrhage is better understood, so that the fear of the important bloodvessels associated with this organ should not be so great. Desjardins says that the removal of the pancreas is not incompatible with life, and that the biliary passages can be reëstablished without undue loss of time. Billroth, in 1894, and Franke, in 1901, have done the only 2 total pancreatectomies, both with success. Neither patient, afterward, presented any troubles due to loss of the organ.

Many partial pancreatectomies have been performed. Of 30 collected cases, there were 9 partial resections of the tail, all but 3 for malignant tumors. There were 2 complete resections of the head, and these alone, Desjardins calls true partial pancreatectomies. There was one in which the body of the pancreas was removed, leaving the head, which avoided sectioning the common bile-duct, and left the tail. This was done for a chronic sclerotic pancreatitis.

Of the 30 cases, there were 16 operative cures and 14 deaths. Of the 2 total pancreatectomies, both were cured; of the resection of the tail, 7 cures and 3 deaths; of the partial of the head, 7 cures and 9 deaths; of the total of the head, 2 deaths. In spite of the good results in Billroth's and Franke's cases, Desjardins does not think that we have a right to do total pancreatectomies, the partial alone being justified by physiology. It is necessary to remove the corresponding part of the duodenum with the head of the pancreas, since both are supplied by the same bloodvessels, they are intimately adherent to each other, and since it is necessary to remove also the retroduodenal lymph glands. The three dangerous zones are: first, where the ascending colon is adherent to the second part of the duodenum. It is necessary here to avoid wounding the colon. The second zone is where the mesenteric arteries emerge from the border of the pancreas, passing in front of the duodenum. The middle colic artery may here be wounded, giving rise to gangrene of the transverse colon. The third danger zone is posterior, where the portal vein and inferior cava are in danger. All three zones can with care be avoided. The careful detachment of the parts is the key to the success of the operation. The head of the pancreas and the duodenum surrounding it are detached en bloc, the duodenum near the pylorus and the beginning of the jejunum being divided between clamps. The common bile duct is divided as near as possible to the duodenum, and the body of the pancreas, including the duct of Wirsung, is divided also. When the removal of the head of the pancreas and the duodenum has been concluded, the pyloric end of the stomach is closed, and the upper cut end of the jejunum is implanted into the stomach, making a gastro-enterostomy. The open ends of the common bile duct and the freed end of the duct of Wirsung are implanted into the upper end of the jejunum, one on one side and the other on the other side. The cut surface of the pancreas is tied off in sections around its duct for hemostasis.

This operation is possible only when the common bile duct and the duct of Wirsung are dilated sufficiently to receive the button of Boari, which is employed to make the connections with the jejunum. In such a case about 30 cm. of the upper end of the jejunum is cut off from the rest, still preserving its attachment to the mesentery. The then upper end of the jejunum is employed for a gastro-enterostomy. The isolated piece of jejunum is anastomosed by one end to the gall-bladder and by the other end to the cut end of the pancreas, hemostasis having been previously effected in this stump. The bile and pancreatic fluid can thus escape only through this new common passage-way made up of the isolated portion of jejunum, which is now anastomosed to the remaining jejunum just below its attachment to the stomach.

The Mesocolic Route: Cholecystojejunostomy, Transmesocolic Gastro-enterostomy.—LOTHEISSEN (*Zentralbl. f. Chir.*, 1907, xxxiv, 605) says that the posterior retrocolic gastro-enterostomy is considered by most surgeons as the normal method. The only objection to it is the occasional occurrence of a vicious circle. Lotheissen has yet to see a failure with it in either v. Hacker's clinic or his own. Brentano has recently shown that the anastomosis between the gall-bladder and the jejunum should be done through the mesocolon. Lotheissen recommended this operation in 1903. The suture is easily carried out after the opening in the gall-bladder has been made. The intestine is tied off with a strip of gauze as in a gastro-enterostomy, and the gall-bladder, after the abdomen is packed off with gauze, is carefully punctured and completely emptied. This permits one to palpate the head of the pancreas and to exclude stones in the common duct. When the operation is completed the gall-bladder lies in the opening of the mesocolon and the visceral situation is normal. The peristaltic movement is unchanged. In doing a gastro-enterostomy, Lotheissen also works through an opening in the mesocolon, in this way doing a posterior gastro-enterostomy. Only once was it necessary to anastomose the jejunum to the anterior wall of the stomach. It is often impossible to bring the transverse colon upward to expose freely the posterior stomach wall. After the operation is completed through the mesocolon, this is closed with a few sutures.

Suppuration of the Hand and Forearm.—KANAVEL (*Zentralbl. f. Chir.*, 1907, xxxiv, 1001) reports the results of experimental and clinical studies on the seat and paths of extension of suppuration in the hand and forearm. The influence of the tendon sheaths was not taken into account. A series of sections was made of the hand and forearm and the connective tissue picked out. Injections were employed to determine the manner and paths of extension along the connective-tissue spaces under varying pressures. Pertinent clinical observations together with the experiments led to the following conclusions:

Extension of the pus from the fingers occurred through the canal for the lumbricalis muscle, to the cavity of the hand and back along the lumbricales, avoiding the tendon sheaths. This was observed clinically and experimentally. Pressure on the hand forced pus in the central connective-tissue space of the hand to the level of the metacarpophalangeal joints, through the lumbrical canals. Extension between the metacarpal bones seldom occurred. Pus passing under the anterior annular ligament to the forearm will be found almost always under the deep flexors of the fingers. In the middle of the arm the pus will almost always be found to be more superficial, lying between the muscles in the neighborhood of the median and ulnar nerve as well as the ulnar artery. It then extends distalward along the course of the ulnar artery, which accounts for the occasional occurrence of secondary hemorrhage.

Palmar abscesses should be evacuated in the course of the lumbrical canals, every infected canal being opened. When the pus originates in the fingers it can be found in the layer over the tendon sheaths of the palm. In protracted cases a large collection will be found in the space under the tendons. In the first group of cases, a simple incision will be sufficient; in the second group a closed forceps may be pushed between the tendons at the upper end of a communicating canal and then the blades opened. If the tendons are not necrotic or the tendon sheaths involved, this procedure will be followed by quick healing. If the thenar space alone is involved, the same method may be employed, passing the forceps from the radial side of the hand. If the central space of the hand is also involved, the forceps should be pushed laterally across and through to the ulnar side. A rubber drainage tube may be left in for a few hours. Rapid healing, with complete cure and return of function, usually results. If the forearm has been invaded the palmar incision may be extended upward through the anterior annular ligament. In a greater number of cases a better result was obtained by lateral incisions in front of the ulna and radius about 3 or 4 cm. above the wrist-joint. The forceps is then pushed across the forearm on the flexor side. The nerves and bloodvessels will be avoided if the forceps is kept close to the bones.

Concerning Ligature of the Common Carotid. A New Method of Ascertaining the Possible Disturbances of the Circulation.—Jordan (*Arch. f. Chir.*, 1907, lxxxiii, 23) says that in 25 per cent. of the cases reported by Pilz, Zimmermann, and Krankepuhl, brain disturbances occurred and 10 per cent. died. Since we cannot ascertain how abundant are the collateral vessels, nor their capacity for dilatation, it is always difficult to determine the prognosis of a ligature of this vessel. To overcome this difficulty Jordan recommends a method which he has tried, successfully, on animals and in one case on man, *i. e.*, the previous, temporary, not firm ligature of the carotid for forty-eight hours. Since the brain symptoms dependent upon a lack of nourishment to the brain occur immediately after the operation, at the end of the first or in the course of the second twenty-four hours; if no trouble from the ligature occurs during this time it is unlikely that there will be any grave consequence from occlusion of the carotid. Experiments on lower animals showed that the ligature did no damage to the intima. Senger, in 1895, employed the temporary ligature for the bloodless extirpation of a tumor of the thigh. The operation should be done under local anesthesia, in order that the effect on the brain may be observed, and the ligature withdrawn on the first appearance of any distinct symptoms. The ligature material may be a small linen band, a rubber band, catgut, or a forceps with its blades guarded with rubber covering. The chief point is to apply the constriction very carefully, until the temporal pulse disappears. A further indication for this procedure is to be found in the suture of arteries.

The Technique of Direct Transfusion of Blood.—Crile (*Annals of Surgery*, 1907, xlvi, 329) bases his remarks on 225 experiments upon animals and 32 clinical cases. The blood was transferred from the artery of the donor to the vein of the recipient. In the clinical transfusions the radial artery of the donor and the proximal end of any superficial vein of the arm of the recipient were employed. Unless contra-indicated, both patients are given a hypodermic injection of morphine twenty minutes before the transfusion begins. The donor is placed on a Trendelenburg table, so that if he should become faint the head may be readily lowered. The recipient is placed upon an operating table with his head in the opposite direction. Infiltration anesthesia of 0.1 per cent. cocaine solution is employed, and the radial artery is exposed. A Crile clamp is applied to the proximal end of the artery and the distal end is ligated; the artery is divided; the adventitia pulled over the free end as far as possible and closely snipped off; a moist sponge now covers this field; 3 or 4 cm. of a superficial vein of the recipient is then likewise freed; the distal part ligated, the proximal closed with a Crile clamp; the distal part is divided with scissors, the adventitia drawn out as far as possible and closely snipped off; the vessels are then inspected and a cannula whose bore is larger than the actual tissue thickness of either vein or artery is selected. The vein may then be pushed through this tube, after which the freed end is turned back like a cuff and snugly tied in the second groove. The artery is then drawn over the vein and tied in the first groove with a small linen ligature. This completes the anastomosis. The clamp is then removed from the vein; afterward, gradually, from the artery; when the blood stream will be seen to pass from the artery across to the vein, dilating the latter. By this method intima comes in contact with intima, the blood is transferred without clotting. The blood lost by the donor is regained in from four to five days, and the amount transferred is under the immediate control of the operator. The rate of transference should be carefully gauged, because of the risk of overcharging the pulmonary circulation.

Ligation of the Ductus Arteriosus.—Munro (*Annals of Surgery*, 1907, xlvi, 335) reports a case of a healthy girl baby, that soon after birth exhibited symptoms of some cardiac lesion, out of keeping with the general appearance of perfect health and development. On severe exertion, such as straining at stool, it would become cyanotic and the cardiac beat would cease. At times the child would apparently die, only to recover as soon as the heart was stimulated with electricity. In a few weeks atelectasis developed, for which artificial expansion of the lungs under negative pressure in a pneumatic cabinet was tried. This met with only temporary success. Auscultation at this time revealed a cardiac murmur with the pulmonary rales, but cyanosis was not a marked feature. After death, which took place without œdema or marked cyanosis, examination showed an open distinct ductus arteriosus, lying easily within reach behind the sternum, without any other defect except a dilated right ventricle. Munro made further dissections and satisfied himself that it would be possible to ligate the duct, provided the diagnosis could be made beforehand. In the newborn the duct of Botalli is a little over 5 mm. in diameter and 10 to 15 mm. long. The length increases generally up to the time of obliteration, which, normally, is complete by the twentieth day. This period of patency is, therefore, normal. Townsend examined 100 newborn babies during the first three days of life, to throw some light on a pathological patency, but he was unable to hear anything instructive. In typical cases the foramen ovale is closed.

Concerning the operation which may be practised on these cases, Munro says that the sternum may be split along its centre with a knife. Retraction of the sternal halves, gives ample room. The right pleural cavity will, probably, be opened, but the left one will not. After retracting the thymus upward, the pericardium is exposed. Its reflection lies so high on the large vessels that the ductus, to all intents and purposes, is intrapericardial. In the upper angle the aorta will be seen on the patient's right and the pulmonary artery on the left. By following close to the aorta toward the under surface of the arch, the ductus, as large as the aorta itself, will be seen as the first vessel to the left, pointing upward and a little to the right. On pushing through the tissues by blunt dissection, the ductus, theoretically, should be easily surrounded by a ligature. Simple crushing might accomplish as much and is worth trying. After closing the anterior pericardial wound the sternum can be sutured or not and the skin closed.

Technique of Bloodvessel Suture.—SWEET (*Annals of Surgery*, 1907, xlvi, 350) believes that the exact end-to-end approximation is easier of execution than other methods; and since it accomplishes that great desideratum, the restoration of tissues as nearly as possible to their original position, it is perhaps theoretically correct. There is a unity of opinions that the suture material should be of fine silk, of a size carefully chosen so that it will completely fill the hole left by the needle, which should be of the smallest possible size, round, either straight or curved, according to the preference of the operator and the site of operation. Experimenters are further agreed that the stitches should include all the coats of the vessel, since the penetration of the intima is a matter of no consequence. All insult to the vessel wall, such as grasping it with forceps, must be rigidly avoided. If the edges of the wound have been crushed they should be refreshened by resecting a bit with a sharp scalpel; since the cut of a scissors is always a crushing one, they should not be used. Three tension sutures of fine silk, impregnated with vaselin, are then laid at equidistant points of the circumference of the vessel ends. An assistant then applies traction to two of these guide sutures in turn, stretching the portion between two sutures into a straight line, facilitating the laying of the continuous suture, and preventing a narrowing of the lumen. If the third tension suture is weighted with a hemostat, the vessel will be arranged in the form of a triangle, and there will be no danger of catching the opposite wall while laying the suture, which is a continuous overhand stitch. The separate stitches should be drawn just tight enough to secure absolute approximation, but not too tight, else the tissues be everted; they must be laid very close together. Absolute asepsis is necessary. Sweet concludes that in any case in which the anastomosis of a bloodvessel is indicated it should be tried; aneurysms and secondary hemorrhages should not occur. If immediate thrombosis occurs, we should be as well off as though the vessel had been ligated; if gradual thrombosis occurs we might well hope that such a process would be more favorable to the formation of a collateral circulation than would immediate ligation.

THERAPEUTICS.

UNDER THE CHARGE OF

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Drugs in Diabetes.—Williamson (*The Practitioner*, lxxix, 1, 136) states that in large doses opium, morphine, and codeine sometimes diminish the sugar excretion in diabetes, more especially in the mild types of the affection; their great disadvantage is their tendency to cause constipation, and as this increases the liability of the patient to coma, these drugs do not appear to be suitable in the aggravated types of diabetes. Heroin also has been tried, but this also, while its use may be beneficial in the less severe forms of the disease, may tend to constipation and occasionally produces toxic symptoms. Sodium salicylate and aspirin are often more satisfactory than opium or its alkaloids, and in mild types of diabetes may induce a diminished sugar excretion, but in the severe forms this benefit seldom occurs, although the general condition of the patient may be improved. The doses should be such as to avoid inducing toxic symptoms, and aspirin is often preferable to the sodium salt on account of its less liability to be followed by ill-effects. Bismuth salicylate is often very useful in instances complicated by diarrhœa, but in the absence of this manifestation it cannot be continued for any considerable period. After giving up treatment by these drugs the amount of sugar in the urine is only too prone to increase again. Nevertheless a certain benefit is to be attained by the administration of these agents. In the presence of diacetic acid in the urine and the imminence of coma the alkalies, particularly sodium bicarbonate, in connection with free purgation by means of mercurials, may cause a disappearance of this dangerous manifestation, but at times this form of treatment is without avail in this regard, especially if the diacetic acid reaction is marked, although the alkaline treatment may be of temporary service in improving the general condition and delaying the onset of coma. The amount of the salt given may be as much as an ounce or more in twenty-four hours, and when premonitory symptoms of coma are present an ounce and a half may be given for several days in succession. When coma has commenced the symptoms may sometimes subside under the influence of large doses of the bicarbonate, but this manifestation is very likely to recur at a later date.

Tuberculin Treatment.—JEANS and SELLARDS (*Johns Hopkins Hosp. Bull.*, 1907, xviii, 195) have studied nine patients treated by means of tuberculin of their own preparation, the treatment having been carried out in accordance with observations of the tuberculo-opsonic indices of the patients in hand. They consider that the study of these nine patients is sufficient to justify the continuation of the method of treatment. In none of the patients has the result been miraculous, but in two, at least, there seemed to be a very definite relationship between the treatment and the improvement of the patient. It is held as important that, in addition to the tuberculin treatment, hygienic measures should be carried out as well. The patients are too few for any general conclusions to be drawn. Other dosage and other interspacing of dosage might have been more effective and perhaps other forms of tuberculin might give still better results. This can be decided only by the clinical results and not, in the author's opinion, by the opsonic curves.

Atoxyl in Syphilis.—UHLENHUTH, HOFFMANN, and ROSCHER (*Deut. med. Woch.*, 1907, 22, 873) have found that injections of atoxyl markedly influence the infection in animals inoculated with syphilis. Their first trials in human beings failed because of too small dosage, but the results of later attempts with larger doses were more successful. One patient in whom the small doses were ineffectual was quickly cured when the amount administered was increased to 7½ minims. In another, syphilis of nine months' duration which had proved refractory to ordinary treatment, nine injections caused a rapid amelioration of the numerous syphilitic ulcers. In a third patient dressings of 10 per cent. atoxyl were followed by a rapid healing of the ulcerations; those which were treated locally healed more quickly than others not dressed, even though injections of the agent were employed at the same time. The injections should be given into the muscles of the thigh; they are painless and cause no local reaction, although they may be followed by anorexia, insomnia, colicky pains and, rarely, by albuminuria. Atoxyl appears to be a true antisyphilitic, especially active early in the disease and in the ulcerations of the early stages.

Transfusion of Blood in Severe Anemia.—MORAWITZ (*Münch. med. Woch.*, 1907, xvi, 767) has employed transfusion of defibrinated blood, about six ounces having been injected at each dose, in six patients. Two of these have been observed too short a time for definite conclusions to be drawn other than that a subjective improvement has taken place. In another patient the treatment was a failure, but in the other three the results were striking. One of these had grown progressively worse under the administration of arsenic, but under the injections of defibrinated blood the blood soon became normal. The other two patients, in whom the prognosis had been unfavorable, improved markedly under the injection treatment. Objective and subjective results show themselves in three or four days after beginning treatment. No serious by-effects were noted.

Thiosinamine in Cardiovascular Fibroses.—RENON (*Revue thérapeutique*, 1907, xiii, 443) has employed injections of thiosinamine on account of its properties in softening cicatricial tissue in instances of cardiovascular fibrosis, valvular affections, and arteriosclerosis. He injects from 15 to 30 minims of a 4 per cent. solution under the skin of the abdomen every two days. The injections are well-borne, only slightly painful, and cause no inflammatory reaction. In mitral lesions the results were *nil*, but in aortic affections a noticeable effect was observed, the dyspnœa being lessened and the arterial tension lowered. The physical signs remained unchanged. In arteriosclerosis the effect was less constant, but in certain patients the dyspnœa became less and the blood pressure was lowered. The action of the drug lasted from three to four months.

The Intravenous Dosage of Digalen.—KOTTMANN (*Correspondenzblatt f. Schweizer Aerzte*, 1907, x, 306) states that this drug is frequently employed in the medical clinic of Berne in patients affected with cardiac insufficiency. Experience has shown the intravenous dosage to be as follows: 15 minims is sufficient to obtain a therapeutic effect, although the usual minimum amount given is five times this quantity. In case the small dose is not followed by definite results the injection may be repeated in a half-hour. The injection is usually followed by a feeling of improvement and an amelioration of the symptoms. Sahli considers that this fact is important from a standpoint of the study of the action of the heart muscle, and is an index as to whether or not digitalis is indicated in a given patient. The injections should be given slowly into the muscular tissue, and it is advisable to induce venous stasis of the part by means of an elastic ligature applied above the site of the injection.

The Three Doses of Digitalin.—HUCHARD (*La quinz. thérap.*, viii, 8, 177) writes of the three methods of prescribing digitalin thus: (1) *The massive dose.* This is given when an anti-asystolic and diuretic action is desired. The amount prescribed is 50 minims of a 1 to 1000 solution, to be given once or twice in a single day. In from thirty-six to forty-eight hours an abundant diuresis is established, with increase in the strength of the cardiac action, relief of the visceral congestion and œdema, and increase in the elimination of the chlorides. If the effect is insufficient the dosage may be repeated in eight or ten days. (2)

The small dose. This is the sedative dose and is given to relieve palpitation and dyspnœa, such as occur in mitral stenosis during the period of compensation. It consists of 5 to 10 drops of the 1 to 1000 solution daily for about five days, every three or four weeks. This quantity of the drug prolongs the diastole. Mitral stenosis is perhaps the only cardiac lesion in which digitalis is indicated during the period of compensation. (3) *The very small dose.* This is the cardiotonic dose; it has no diuretic or true cardiac action, and may be continued for weeks or months with intermissions at intervals of from fourteen to twenty-one days. The proper amount is 3 to 4 minims of the 1 to 1000 solution; this dosage is unlikely to cause more than very mild toxic symptoms, and these occur only because of the slowness of the elimination of the drug. The digitalin solution is given hypodermatically, or tablets of equivalent strength may be substituted if it is desired to employ oral administration.

Veronal as a Hypnotic.—Foxwell (*Folia Therap.*, 1907, i, 69) in a comparative study of drugs of this class voices the statement that veronal is, taken altogether, our most satisfactory hypnotic. Its action is sure, powerful, and not accompanied by untoward by-effects. Rarely giddiness, headache, vomiting, and transient eruptions have been noted, but no serious complications. As a simple hypnotic 3 grains is a sufficient initial dose; seldom, except in mental patients, will more than 7 or 8 grains be required. Cumulative action is likely to occur, but there is probably no danger of any sudden *dénouement,* though there may be an increasing effect from continuance of its administration in the same dosage. The drug is an excellent sedative in doses of 1½ to 2 grains thrice daily. As a hypnotic, given in hot water, milk, or tea, it usually induces sleep in a half-hour. The slumber is quiet and natural and, if the dose has been small, no unpleasant heaviness is experienced on awakening.

Aspirin and Sodium Salicylate in Rheumatic Conditions.—Roch, Jeanneret, and Lamuniere (*Therap. Monats.*, 1907, xxi, 239), after a comparative study of these two drugs, draw the following conclusions: To those who know how sensitively rheumatic patients react to sudden diminution of the dosage of sodium salicylate, it seems strange that one can without untoward effect change suddenly from doses of 75, 90, or 100 grains of sodium salicylate to 45 grains of aspirin. The latter has fewer unpleasant effects upon the alimentary tract than the sodium salt, and is superior to it in the treatment of obstinate subacute rheumatism, even when exacerbations are likely to take place. The action of aspirin is explainable on the theory that while it is transformed in the intestine and its elimination product is identical with that of sodium salicylate, it is absorbed more slowly and with greater regularity than is the latter substance, and the organism consequently becomes more saturated with the salicylic acid. Over and under saturation are much more easily controlled in treatment with aspirin than in treatment with sodium salicylate. It seems probable that the nervous symptoms, headache, tinnitus, delirium, etc., which are not uncommon results of the administration of the salicylates, may be avoided when aspirin is employed, for the reason that the absorption of this drug is so continuous and regular.

The Standardization of Cardiac Remedies.—EDMUNDS (*Jour. Amer. Med. Assoc.*, 1907, xlviii, 1744) draws certain conclusions upon this subject, among which are the following: The necessity for a uniform strength of the cardiac tonics upon the market is undeniable, but how this is to be obtained is another matter. If the tinctures are standardized those of an individual firm may be uniform, although this does not necessarily follow; but each firm having its own standard, there can be little more uniformity than at present. What is needed is a national standard such as the Pharmacopœia furnishes for chemical assays. As the matter now stands the physiological assay is made by the physician at the bedside. He gives the drug for a reasonable time and gets no effect perhaps, and then has to increase the dose until the desired action is attained. No amount of standardization would ever do away with his ascertaining the physiological dose for the individual patient, but it certainly would shorten the process and avoid the loss of much valuable time, which may mean the life or death of the patient. [If the mandates of the Pharmacopœia are obeyed any manufacturer can produce uniform and official preparations.—R. W. W.]

PEDIATRICS.

UNDER THE CHARGE OF

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The Treatment of Some Common Tuberculous Affections in Children.—C. R. KEYSER (*British Journal of Children's Diseases*, 1907, iv, 277–281) finds "subcutaneous tuberculous foci" quite commonly in children. They form small, round masses in the subcutaneous tissues, and are freely movable under the skin and the deeper structures; occasionally they occur in the deep cellular tissue. Later the skin becomes reddened, tender, and still later growth and skin adhere and the tuberculous mass breaks down. They are most common on the limbs, but have nothing to do with disease of the bone; they are precisely similar to gummas forming in the same situation. The treatment should not consist of incision and drainage, as the "abscess" does not contain a drop of true pus. Very often, if this is done, pyogenic organisms are admitted into the cavity and true pus forms.

The treatment he advises is incision and complete evacuation of all the contents, most particular attention being taken to remove all the semisolid caseous masses, and to scrape away the so-called pyogenic membrane. The scrapings, etc., should be removed with normal salt irrigation, the cavity filled with iodoform emulsion and the wound accurately sutured and pressure applied. Re-accumulation should be treated by aspiration and pressure. He treats tuberculous osteomyelitis by laying the bone freely open, scraping away all the tuberculous matter, filling the cavity with decalcified bone chips, suturing the wound, and applying a splint.

A Case of Morbus Cordis.—T. R. Wipham (*British Journal of Children's Diseases*, 1907, iv, 252) reports a case of heart disease, in which there had been 7,800,000 red blood corpuscles per c.mm. The patient was a girl, aged eleven years, who was subject to frequent attacks of epistaxis. The heart was much enlarged, and there was a thrill at the apex and at the base, especially over the aortic area. Presystolic and systolic murmurs were present at the apex, and the latter was loud and rough at the base, especially to the right of the sternum. The second sound occasionally could be heard in the pulmonary region, but only faintly; elsewhere, it was entirely absent. There was no cyanosis or clubbing of the fingers. The patient had recently had acute rheumatism, but there was no history of a previous attack or of chorea. The diagnosis suggested was mitral stenosis and possibly a congenital narrowing or coarctation of the aorta.

Tuberculous Disease of the Spine in Children.—Olive Elgood (*British Journal of Children's Diseases*, 1907, iv, 229–252) considers spinal caries an inaccurate name to give to all forms of tuberculous disease of the vertebræ. A primary, superficial caries does occur fairly frequently in adult life, but in children this type is rare. The disease conforms primarily to one of two pathological types: (*a*) The localized soft, caseous deposit, or the sequestrum, in the interior of the bone, which is the usual form met with in children; (*b*) the primary superficial caries. In the former tubercle bacilli become deposited from the blood in the cancelli of the vertebræ, and give rise to a collection of epithelioid cells, which either become differentiated into round areas forming microscopic or macroscopic tubercles, or remain undifferentiated as diffuse, infiltrating tuberculous tissue. In either case an area of simple inflammatory round cells occurs round the specific formation due to the irritation set up, primarily by the pressure of the latter in the surrounding normal tissue, and secondarily to the gross weight of the trunk above the seat of the disease pressing on the softened vertebræ. As the tuberculous disease spreads into and replaces the granulation tissue, the latter extends into the normal bone, progressively absorbing it, while in the central part the tuberculous tissue either breaks down into caseous pus forming the soft tuberculous deposit, or may, in parts, caseate and in other parts organize into fibrous tissue and sclerosed bone, which then comes to lie in caseous pus, forming sequestra; the latter are not frequently seen in children. The disease ultimately reaches the surface, either at the front or sides of the body, or it perforates backward into the spinal canal. After reaching the surface of the body it may continue as a secondary superficial caries, spreading along the surface of the bodies and destroying the intervertebral disks, or it may form a localized abscess, surrounding the spot where the caseous pus has reached the surface.

Primary superficial caries is due either to a primary deposit of tubercle in the periosteum or surface of the bone, or is the secondary result of the perforation of an internal deposit on to the surface. The disease being on the surface spreads rapidly to the adjacent soft parts, giving rise to the typical tuberculous abscesses of the various regions. In rare cases the disease occurs in the transverse processes, or spines, and then quickly gives rise to an abscess, the disease being so near the surface.

Spinal caries is not so common as hip disease in children, the former making up 27 per cent., the latter 39 per cent. of the cases seen by Elgood. Traumatism preceding the caries was found in but 14 per cent. of Elgood's cases. Girls made up 33 per cent. of her cases, and the resisting power of their blood to bacterial invasion of bones was invariably greater than in boys. A family history of tuberculosis was present in 6 of her cases. From the standpoint of clinical signs the author makes the following classification: (1) Early cases before the establishment of curvature, only diagnosticated by the determination of the presence of rigidity of the spine. (2) Cases with the establishment of spinal deformity, but without complication. (3) Cases with or without deformity, but with complication: (*a*) abscess; (*b*) some concomitant tuberculous trouble, such as tuberculosis of the lungs or glands, joint and bone affections; (*c*) sepsis; (*d*) compression of the spinal cord.

The treatment is classified under the following headings: (*a*) *Conservative methods:* This means rest and is suitable in the very early and uncomplicated cases. Supine position in bed with extension to each leg and counterextension to the head in cervical disease cause the pain to cease within twenty-four hours; the patients are kept thus for six weeks after the last complaint of pain. This is followed by a plaster-of-Paris jacket, and in upper-spine cases a jury-mast is incorporated in the layers. The patient is kept on his back for one year after application of this support, and permitted to move sparingly the next year. Weakness of the back, thereafter, is best met with slight kneading massage of the muscles of the back, combined with a light support. (*b*) *Operative procedures:* The removal of pus under the strictest aseptic precautions; this is not curative. Laminectomy for the purpose of removing the whole area of the disease; 1 patient thus treated died of meningitis, 2 others recovered. Laminectomy for the purpose of reducing deformity; this should only be performed in the late stages of quiescence of the disease, and should be very carefully considered, before being undertaken, as it may easily light the process up again. (*c*) *Vaccine treatment:* This can only be an adjunct to one of the former methods described. Injections of tuberculin can be combined advantageously with any of the methods.

OBSTETRICS.

UNDER THE CHARGE OF

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Advanced Ectopic Gestation.—SITTNER (*Archiv f. Gyn.*, 1907, Band lxxiv, Heft 3) reports 2 cases of abdominal pregnancy. One was that of a woman pregnant for the fourth time, having an indefinite swelling to the right of the uterus. Appendicitis was suspected, and pain and collapse occurring, led to a diagnosis of ruptured ectopic gestation. The patient was not subjected to operation, and the uterus continued gradually to enlarge. At about the seventh month abdominal section was performed, and abdominal pregnancy was found, the placenta being attached to the intestine over the right pelvic region. Sittner calls attention to the difficulty of diagnosis in cases of abdominal pregnancy, which is frequently not made before operation. In 77 cases of ectopic gestation in Schauta's clinic, the diagnosis of an inflammatory condition in the Fallopian tube was made in 7. When the uterus ruptures in early pregnancy the diagnosis is especially difficult.

When the child is living, operation gives a high mortality, which is gradually lessened after the child dies. The mortality of ectopic gestation without operation is stated as 68 per cent., and 23 per cent. in those operated upon. The death of the fœtus does not entirely do away with danger from placental hemorrhage, as the placental vessels are not completely obliterated for six months after the death of the fœtus. If the diagnosis can be made, it seems probable that early operation while the child lives is least dangerous for the mother. The mortality among children born after ectopic gestation is, during the first twenty-four hours, nineteen times greater than in cases of entopic pregnancy. These children are ill-developed, and but about 50 per cent. of them reach adult life. Delay in operation is dangerous for the mother, from peritonitis, diseases of the bladder, emaciation, and rupture of the sac. While the complete removal of the placenta has been considered exceedingly dangerous, in 107 cases of ectopic gestation the placenta and membranes were removed in 53; in 54 cases the placenta was left; when it was removed, the mortality was 18.8 per cent.; while in cases in which the placenta was allowed to remain the mortality rose to 57.4 per cent.

Among the difficulties experienced in removing the placenta, in 10 per cent. extensive adhesions were present; the placenta was situated within the folds of the broad ligament in 25 per cent., and attached in an atypical position in the pelvis in 37 per cent.

Sittner's paper is based upon 126 cases in which section was done after the tweny-first week of pregnancy, with a living child. The literature on the subject shows 14 cases in which one operator has twice operated for abdominal pregnancy at full term with a living child. In only 4 instances has the same operator had 3 such cases. Sittner believes that most cases of secondary abdominal pregnancy develop through actual rupture of the muscular tissue of the Fallopian tube, occurring after erosion. This develops gradually, and tends to explain those cases of abdominal pregnancy without symptoms of tubal rupture. When rupture is sudden as the result of violence, the sac of the ovum is loosened and pregnancy ceases to progress.

The Lymphatic Glands in Pregnancy.—Meyer (*Surg., Gyn., and Obstet.*, May, 1907) has studied 59 cases in the obstetric wards of the Johns Hopkins Hospital to determine the condition of the lymphatic glands. These patients were palpated weekly from two to twelve weeks. The greater number of examinations were made before labor. The age of the patients varied from sixteen to forty years. About

25 per cent. were between sixteen and twenty years, 65 per cent. between twenty and thirty, and 10 per cent. between thirty and forty years; 30 of the patients were white, 29 negroes.

For examination, the patient was placed in bed, with little clothing, to secure muscular relaxation and to give the best possible examination. To make the examination as nearly accurate as possible, they were made on the same day of the week and at the same hour in the day. Nine o'clock in the morning was the time chosen. An especial effort was made by palpating on the third day after labor to ascertain the effect produced by lactation upon the axillary and infraclavicular lymphatic glands. Most of the patients were examined but once after labor, some twice, and a few more frequently. When a pelvic infection like tuberculosis was present, the cases were excluded. All of the accessible lymphatic glands were examined except the popliteal. In the axillary it was noted whether the palpable glands were in the pectoral regions at the bottom of the axilla or in both regions. It was found useless to attempt to count definitely the number of glands in each region or group. The main question was to ascertain the size of the glands and, as far as possible, their consistence. Examinations were made independently of the patient's previous history. The patient, however, was questioned concerning the symptoms of tuberculosis of the glands, affections of the breasts, skin, teeth, scalp, sore throat, tonsillitis, and venereal disease.

The following is a summary of the results obtained: The total number of cases being 59, in but 1 of these patients could no glands be found upon examination. In none of the patients were the glands palpable in all the regions examined; in 98.3 per cent. of the cases, at some time the axillary glands could be distinguished; in the same percentage of cases the inguinal glands were also palpable at some time; in 74.6 per cent. the submaxillary glands could sometimes be felt; in 66.1 per cent. the axillary glands could be distinguished at every examination; in 62.7 per cent. the inguinal glands; in 44.2 per cent. the submaxillary glands; the supratrochlear were palpable in 30.5 per cent.; the posterior auricular in 10.1 per cent.; the superficial cervical glands in 5 per cent.; the suprahyoid in 1.7 per cent.; a progressive enlargement of the axillary glands was observed in 25.4 per cent.; in the inguinal 15.2 per cent.

Lactation caused enlargement of the axillary glands in 62.5 per cent. These glands could not be palpated in the second week of the puerperal period in one patient. Two weeks before delivery in one patient the axillary glands were enlarged and tender. After delivery the inguinal glands decreased in size in 21.6 per cent.

It was interesting to observe that the lymphatics were not symmetrical in the two sides of the body. The differences were greatest in the axillary, submaxillary, and supratrochear glands, and much less in the inguinal region. There were two patients in whom the glands could not be palpated.

The first was a white woman, aged twenty-nine years, the mother of five children. Her last labor was complicated by manual removal of the placenta. She had had children's diseases and an abortion, and the placenta had been manually removed at previous labors. This patient was under observation for seventeen days only. On two examinations no glands could be palpated.

The second case was that of a young negress aged sixteen years, rachitic, in whom no glands could be palpated on two successive examinations. During the third the right axillary glands were distinct, the inguinal glands barely perceptible. At the fourth examination the axillary and inguinal glands of both sides were palpable, but small. The axillary glands could not be detected sixteen days after labor, although the mother nursed her child. Four weeks after labor the inguinal glands of the right side could not be observed. The patient was delivered by pubiotomy, and the inguinal glands of the left side on which the operation was done were easily palpable. Engorged breasts invariably cause tenderness of the axillary glands, either before or after labor. Affections of the teeth or throat cause enlargement of the submaxillary glands. It could not be definitely decided whether the enlargement of the axillary lymphatics was due primarily to changes in the mammary glands or to pregnancy itself. In one case in which lobules of the mammary gland were found in the axilla, engorgement of the breasts was accompanied by sudden enlargement in the lymphatics. These glands decreased in size as the breasts grew smaller. Very often when lactation was developed the pectoral glands were strikingly enlarged, while those in the depths of the axilla were only slightly affected. It seems probable that the lymphatic glands of the whole body must be affected by pregnancy, but these changes are slight, gradual, and often transient, and enlargement which can be clearly detected seems to be caused by local conditions resulting from pregnancy.

The changes in the inguinal glands seem to be based primarily upon the results of pressure from the lower abdomen and pelvis. Age and multiparity seem to have no influence upon the lymphatic glands. Leucorrhœa before labor, injury at birth, obstetric operations, fever in the puerperal period, but not necessarily, produce changes in the lymphatics. In 1 case in which the child had ophthalmia, and the mother a well-marked leucorrhœa, the inguinal glands were enlarged three days after labor. This enlargement was temporary only, and soon disappeared.

We may explain the fact that the inguinal glands were not enlarged in patients that had tears in the perineum and vagina by aseptic and antiseptic methods employed at labor. It was observed that the inguinal glands were always more firm and flat than the axillary; the submaxillary were oval in shape and hard. The inguinal glands, however, undergo an early sclerosis, and possibly the changes in these glands had resulted from a previous venereal infection.

A comparison of the races shows that in the negress the lymphatic glands were larger in every region where they could be palpated. Progressive enlargement of the axillary glands was noted in 30 per cent. among negresses, and in 17 per cent. among the whites. These glands increased with beginning lactation more frequently among the negresses than the whites.

In giving his summary and conclusions, Meyer states the points already noted. He finds that no general enlargement of the lymphatic glands was observed in pregnancy. The enlargement noticed in the maxillary and submaxillary regions was due to local causes in the axilla and groin, induced by pregnancy; in the case of the submaxillary glands to the lesions of the teeth, gums, mouth, and throat. These differences are thought to depend on the fact that the breasts were brought into active function, while the mouth, teeth, and scalp are very frequently neglected and the site of pathological conditions. In none of these cases could it be observed that new lymphatics developed during pregnancy.

GYNECOLOGY.

UNDER THE CHARGE OF

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The Histological and Clinical Significance of Malignant Chorioepithelioma.—SCHMAUCH (*Surg.*, *Gyn.*, and *Obst.*, 1907, v, 259) considers this subject very fully and declares that not always is chorioepithelioma malignant. Polano has expressed the belief, after a very careful analysis of all the cases of this disease in which successful operations had been performed, that 50 per cent. of the patients would have recovered without operation. Schmauch believes that every case of so-called surgical cure would have recovered spontaneously. He sums up as follows: The chorioepithelial proliferations described in literature as chorioepithelioma malignum have nothing in common with a malignant growth, as generally interpreted. On the other hand, the whole doctrine of chorioepithelioma proves a great support of Ribbert's theory of the development of carcinoma. Epithelial cell complexes, torn from their origin and deported, proliferate and form a kind of autonomous growth. There is only a gradual difference between the simple invasion of the uterine muscles by villi and their sequels, such as adherent placenta, the common and destructive placentary polyp, and the common and destructive mole; between the localized chorioepithelial growths, limited to the uterus and the placental region, and the villous infarcts, developed by deportation of villi; and finally between those cases in which the lungs are able to retain and master the scattered fœtal cells (mostly syncytial and atypical forms), and the typical chorioepithelioma malignum, which becomes generalized through general metastases. Only the latter two types deserve the name of malignant chorioepithelioma.

In recent publications concerning chorioepitheliomatous proliferations in the male, a tendency is to be noticed to regard those cases of generalized chorioepithelioma, in which the uterus and adnexa proved free from growth, as of teratoid genesis. However, as the connection with pregnancy is an established fact, we have no right to hunt for complicated theories. In many of the cases, in which the uterus was free from growth, a closer examination probably would have revealed smaller neoplastic foci in the parametrium. All the enumerated conditions are sequels of pregnancy. We deal with proliferations that trespass the physiological limits necessary for the development of the fertilized ovum and therefore become pathological. Fœtal cells and villi, which normally invade the uterine mucosa to bring about fixation of the ovum and further development, pervade the musculature of the uterus, become deported into vaginal veins, and finally lead to the death of the bearer by general dissemination. Between the physiological embolism of placentary cells and the typical malignant chorioepithelioma there is only a gradual difference. Schmorl states expressly that small metastatic foci from a malignant chorioepithelioma cannot be differentiated microscopically from chorioepithelial embolism found in a vesicular mole.

Whether a badly nourished inferior mucosa facilitates the invasion of the villi, and leads to adherent placenta and placentary polyp or not, is mere speculation. Whenever villi have invaded intramuscular bloodvessels (not intravillous spaces), a deportation of villi or larger placentary cell complexes is rendered easy. When retrograde deportation in the external genitalia takes place, it will become manifest to the naked eye, and when deported into the lungs bloody expectoration will result. It is very probable that a number of the small-sized hematomas of the vulva and vagina, the so-called "thrombus vaginæ," are caused by villous infarcts. Older German text-books of obstetrics, for instance that of Spiegelberg, speak of such hematomas appearing during pregnancy, and some days after labor. While the development during labor was easily understood, in the other forms a friability of the bloodvessels was supposed to be the cause. It sounds strange to read reports of such patients, and hear of the protracted convalescences and of the grave alteration of the general conditions.

The so-called vaginal metastases, appearing after an operation, from which the patient recovers completely, seem at first sight absolutely malignant. They become easily understood, however, when one assumes that they were already deposited at the time of the operation, but did not become manifest until the insult, connected with the operative interference, led to the formation of a hematoma. The presence of villi in a villous infarct is not absolutely necessary for that diagnosis. They might become absorbed, or at other times it is possible that they will not be found. Among 11 cases classified by me as villous infarcts, villi were present 8 times.

When chorioepithelioma is regarded as a cellular injection and not as a true neoplasm, it will be useless to search for histological marks, proving malignancy or benignity. As the typical as well as the syncytial form may present all the different stages, we will never be able to render a definite opinion upon the eventful malignancy of the process from an extirpated vaginal nodule or uterus. The prognosis should always be guarded. As in bacterial infection, the seriousness of the invasion will be judged by the general, clinical symptoms, and not so much by the appearance of the diseased part of the body.

As the results become evident from the tabulation of the cases, only a few facts will be mentioned here: (1) The malignancy of the generalized chorioepithelioma and the prevalence of the typical chorioepithelioma among this class. (2) The lesser malignancy of the transition form syncytial chorioepithelioma (predominance of large syncytial cells besides Langhans' cells and syncytium). (3) The harmlessness of the atypical chorioepithelioma. Among the 206 tabulated cases, I find 10 which I regard as atypical chorioepithelioma consisting of large cells only, without admixture of Langhans' cells and syncytium. 14 of them recovered after operation and 5 died. In 2 of these 5, sepsis is given as cause of death, in 1 nephritis, and in another one death soon followed the operation, while in the fifth the cause of death is not stated. There is so far not one case of generalization in an atypical chorioepithelioma reported. (4) The comparative benignity of all those cases in which villi were found in the extirpated uterus or in the vaginal infarcts. (5) While 43 cases of chorioepithelioma that proved to be malignant were preceded by abortion, only 24 followed molar pregnancy. Again, while 39 operated and recovered cases were preceded by mole, only 11 cases recovered after labor at term. On the other hand, 30 cases are recorded that died of malignant chorioepithelioma following full-term labor. Herewith is proved the frequency of chorioepithelial proliferation after mole and the danger of sepsis and exsanguination in destructive mole and placentary polyp, but not the histological malignancy of such relatively frequent proliferations. (6) The great importance of an exact anamnesis in judging the histological dignity of proliferations of the cells forming the cell-cover of villi.

Schmauch expresses great regret that gynecologists have considered the histology of so much greater importance than the symptomatology, which Schmauch considers should be reversed.

Postoperative Thrombosis and Embolism.—MILLER (*Amer. Jour. Obst.*, 1907, lvi, 347) classes the causes of postoperative thrombosis and embolism as follows: (1) Inflammatory thrombi, which are produced through acute or chronic inflammation of the wall of the heart or bloodvessels. As a consequence of the inflammation, the wall is thickened and the endothelium injured. (2) Traumatic thrombi produced through injuries to the vessel wall. To this classification belong those thrombi caused by ligation, severing, or tearing of the vessel. (3) Compression or dilatation thrombi, which are caused by slowing or stagnation of the blood stream. The compression can arise by the pressure of tumors, exudates, etc., and the dilatation may be due to actual disease, loss of elasticity, or excessive thinning of the vessel wall. Certain cardiac thrombi probably come under this head. (4) Marantic thrombi, which are due to diseased conditions of the blood, degeneration or weakness of the heart, and slowing of the blood stream.

Miller refers to the interesting experiments of Carrel and Guthrie on the anastomosis of bloodvessels and the transplantation of viscera as strongly indicating that infection and injury to the vessel wall are of primary importance etiologically. They found that in animals operated upon, in which the technique was rigid and the endothelium of the bloodvessels accurately adapted, thrombosis occurred but rarely. This does not explain the predilection of certain vessels to produce thrombi. Probably all the causes mentioned enter into most cases. Certainly they are most commonly formed when operations have been done for myoma or carcinoma of the uterus or ovarian tumors of prodigious proportions. Miller considers that the abnormal blood conditions notoriously present in these cases are the chief causes in them. He also lays stress upon the enlargement or dilatations of bloodvessels resulting from tumor pressure, as being a potent etiological factor, and he refers to the long oblique course of the iliac vein across the pelvis behind the rectum and sigmoid flexure and the left iliac artery, as an explanation of the great frequency of involvement in thrombosis after operation. His paper is terminated by a statistical statement of the comparative frequency of involvement of various regional areas and structures.

Multiple Ovarian Cyst Successfully Removed from an Infant Aged Eleven Months.—C. W. MacGillivray (*Lancet*, 1907, i, 1487) reports the case of a girl of eleven months, whose mother, previous to the patient's birth, had 5 miscarriages. At the age of three months the child's abdomen was seen to be hard and large; at intervals the child suffered marked pain and had diarrhœal attacks. The abdomen increased in size rapidly and the child became more and more emaciated. Examination showed the child to be cyanosed and to have difficulty in breathing; the abdomen was enormously enlarged, the abdominal walls thin, and the umbilicus projected. The entire abdomen was dull to percussion. A puncture brought forth clear, yellowish fluid, and the relieved surface was found to be irregular and nodular. Operation revealed the condition to have been caused by a multilocular ovarian cyst attached at three points: below the liver, about the level of the umbilicus, and near the brim of the pelvis on the right side. These points contained large veins and arteries. The cyst was made up of two halves about equal in size and attached to each other by loose connective tissue. The length of the tumor was eleven inches, the thickness five. It filled up the whole distended abdominal cavity from the diaphragm to the true pelvis. The child recovered uneventfully.

Tubal Pregnancy Associated with an Ovarian Cyst.—E. G. E. Arnold (*Lancet*, 1907, i, 1490) reports the case of a woman upon whom he performed a laparotomy because of an ectopic gestation which was on the point of rupture; at the same time he found and removed an ovarian cyst the size of a tennis ball from the other side. The right tube and left ovary both appeared healthy and were therefore not disturbed. This tumor could not be found at an examination made nine months previously, and it has to be assumed therefore that it originated or at least grew very rapidly under the influence of the pregnancy.

Bacteriological and Clinical Examinations to Determine the Value of Modern Procedures in Performing Aseptic Gynecological Laparotomies. A large number of such examinations lead W. Hannes (*Zeit. f. Geburt. u. Gyn.*, 1907, lx, 1) to formulate the following conclusions. (1) Wounds produced during operations are constantly threatened with bacterial invasion from organisms on the hands of the operator and his assistants, from organisms on the skin of the patient, and from organisms floating in the air of the operating room. (2) Wearing dry, sterile rubber gloves on the disinfected dry hands makes the transference of bacteria from the hands of the surgeon completely impossible; this has been demonstrated repeatedly. (3) In a large number of instances, particularly in cases in which the operation lasts but a short time, it is unquestionably possible to keep the abdominal wall, which cannot be rendered absolutely aseptic, from coming in contact with the operative surface. Gaudanin is much more suitable for this purpose than rubber dam; it particularly has many advantages over rubber cloth in those cases in which considerable fluid is constantly flowing over the abdominal coverings. (4) The possibility of an occasional infection from the air of the operating room is made likely by the result of bacteriological examinations. (5) Operative wounds known to contain bacteria may heal perfectly and promptly and do so very often. (6) When the technique is carried out rigidly even to the smallest details, the possibilities of errors and dangers is markedly lessened. (7) The use of a weak disinfecting fluid instead of the questionable normal saline solution for the purpose of rinsing the gloves seems an advantage; it were also better if the sterile instruments were kept in an antiseptic solution to protect them against the organisms in the air of the room. (8) The sterilizing apparatus should be examined bacteriologically from time to time. (9) Even when the most rigid technique thus far described is employed, it is impossible to assert, as has been done repeatedly within the last few years, that an absolutely germ-free operation can be performed.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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Eruptive Sore Throat.—EDMUND COCKS (*Jour. Amer. Med. Assoc.*, November 24, 1906) recently reported a case of pemphigus limited to the mucous membranes. The patient was a nervous woman, aged thirty-seven years, in the seventh month of pregnancy. For some months deglutition had been painful at the menstrual periods in consequence of numerous pea-sized vesicles on the tongue, buccal mucous membrane, pharynx, and lips. When seen by Dr. Cocks there was not a pea-size space of tongue, lips, buccal mucous membrane, pharynx or rhinopharynx that was not the site of vesicles. Vesicles were found on the conjunctiva, in the rectum, and in the vagina. The cervix was free. The patient died three weeks after confinement. Vesicles were found postmortem in the nares, mouth, tongue, pharynx, œsophagus, stomach, and intestinal tract from the pylorus to the anus. There were a few disseminated vesicles on the serous surface of the liver, spleen, intestines, and pleura. Cocks presumes the case to have been one of pemphigus limited to the mucous membranes, probably due to indican, the result of intestinal fermentation of several years' standing.

Hyperkeratosis of the Tonsils.—GEO. B. WOOD. (*Annals of Otol., Rhin. and Laryng.* December, 1906) discusses and illustrates the etiology of hyperkeratosis of the tonsil, and places particular emphasis upon the importance of disassociating the leptothrix and other microörganisms as the causal factor.

Aberrant Tonsil.—CHAUVEAU (*Arch. internat. de laryng., d'otol., et de rhin.*, November and December, 1906) reports and pictures an aberrant hypertrophied tonsil behind the right posterior palatine fold in a man twenty-nine years of age, who had been sensible of discomfort for a few days only.

Resorption of Lymphoid Vegetations at the Vault of the Pharynx under the Influence of Infectious Disease.—To the few cases on record CHAUVEAU (*Arch. internat. de laryng., d'otol., et de rhin.*, November and December, 1906) adds an observation of rapid regression of adenoid vegetations in a case of measles in a child ten years of age; and he refers to three other cases of rapid regression under the influence of influenza, reported by him in *La france médicale*, September 1, 1899.

Fatal Syphilis of the Rhinopharynx.—FISCHENICH (*Verhand. des Verein Süddeut. Laryng.*, 1906) reports a case of syphilis of the rhinopharynx with fatal termination from repeated hemorrhages, in a man, forty years of age, who had acquired the initial disease some seven years previously.

Tuberculous Growth of the Pharynx.—SCHILLING (*Arch. internat. de laryng., d'otol., et de rhin.*, November and December, 1906) reports a tuberculous tumor at the vault of the pharynx, in a man fifty years of age, without tuberculosis in any other region. It was removed with an incandescent snare, and the remnants were destroyed with caustics. The patient had no trouble after the ablation of the growth.

Myxosarcoma of the Pharynx.—CALDEIRA CABRAL (*Arch. internat. de laryng., d'otol., et de rhin.*, September and October, 1906) reports the case of complete removal of adenoid vegetations from the pharynx of a young mulatto girl, ten years of age, who two months later presented similar symptoms with the addition of trismus and dysphasia.

Vegetations were felt behind the palate, and were removed by curetting. Histological examination revealed myxosarcoma. Recurrence ensued within ten days.

Extreme Congenital Stricture of the Œsophagus Successfully Relieved under Œsophagoscopy.—GUISEZ (*Annal. des maladies de l'oreille, du larynx, du nez, et du pharynx*, December, 1906) reports, among other cases, a remarkable one of extreme congenital stricture in a young man, eighteen years of age, who had never been able to take any nourishment but milk and water. The patient was being nourished through a gastric fistule which had been made for that purpose a short time previously. Under œsophagoscopy a large diverticulum at the inferior third of the œsophagus was found filled with mucus and curdled milk which, on aspiration with a pump amounted to a half litre. At the inferior portion of this pouch a sort of semilunar valve was observed obstructing the calibre of the œsophagus almost completely, and leaving but a very small orifice upon one side. This orifice barely admitted the smallest œsophageal bougie in the doctor's equipment.

In displacing the œsophageal tube toward the left, a sort of blind cul-de-sac of the left wall of the œsophagus was seen in which the bougies were always caught in attempts at dilatation. Two months later this membrane was incised under œsophagoscopy after having been painted with a solution of adrenalin 1 to 1000, and then dilated with bougies of 4 and 6 mm., respectively. The patient was allowed to drink milk and gradually became able to take solids, so that at the end of three months he was able to eat any food, and had gained three pounds in weight. At the time of the report a bougie of 9 mm. could be passed.

Tuberculoma of the Nasal Septum.—A. Onodi (*Arch. internat. de laryng., d'otol., et de rhin.*, November and December, 1906) reports a case of presumptive malignant growth of the nasal septum in a man sixty years of age. Thorough radical removal of the diseased septum and some adjacent structures was practised by external access, and the patient had an excellent recovery. Microscopic examination of the parts removed revealed tuberculoma of the nasal septum.

Tracheotomy Cannula in a Bronchus.—The dropping of an ill-cared-for tracheal cannula from electrolytic corrosion at the points of solder, where the contact of two metals and the corrosive liquid supply the conditions, has not been infrequent, especially before the practice of aseptic surgery. A recent case reported by M. Jaboulay (*Annales des maladies de l'oreille, du larynx, du nez, et du pharynx*, November, 1906), is illustrated with an unusually distinct *x*-ray reproduction showing the corroded cannula in the right bronchus. The foreign body was eventually removed under intra-oral bronchoscopy after failure of other methods, including resection of a rib; but, unfortunately, the patient died twelve days later of bronchopneumonia.

Gumma at the Bifurcation of the Bronchi.—J. Garel (*Annales des maladies de l'oreille, du larynx, du nez, et du pharynx*, July, 1906) reports a case of gumma at the bifurcation of the bronchi detected by bronchoscopy. A characteristic ulceration of the superior upper border of the left Eustachian tube, which had not given any trouble whatever, had indicated the existence of syphilis, and the patient's cavernous, barking cough pointed to bifurcation or stenosis of the air passage, and thus led to the inspection by bronchoscopy.

Carcinoma of the Tongue and Larynx.—In reporting a case of modified transhyoid pharyngotomy with extirpation of the epiglottis, the upper third of the larynx, and the posterior two-thirds of the tongue, Ricardo Botey (*Archives internationales de laryngologie, d'otologie, et de rhinologie*, March and April, 1907) extols his modification of the procedure, which includes the partial resection of the hyoid bone and deep suturing of the tissues. The patient was a man, fifty-two years of age, and the morbid growth was found to be a squamous epithelioma. The extirpation was preceded by a tracheotomy six days previously. The main operation occupied more than two hours, but was not followed by shock and the patient made an excellent recovery, being apparently well with an intelligible speech and facile deglutition a month or so afterward.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
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The Presence of Meningococcus and Micrococcus Catarrhalis in the Nasopharynx and Disinfection by Pyocyanase in these Infections.—Emmerich and Löw have made many observations upon the bactericidal action of pyocyanase toward such organisms as the cholera vibrio and diphtheria and typhoid bacilli, and they believe that the active principle of pyocyanase is a bacterolytic enzyme. Zucker has recently made use of pyocyanase as a local disinfectant in diphteria. Jehle (*Wien. klin. Woch.*, 1907, xx, 8) finds that pyocyanase in dilute solutions is capable of inhibiting or destroying the growth of meningococci and of Micrococcus catarrhalis. It is much more active when added to pure cultures of these organisms than to exudates containing the cocci; for in the exudates many of the cocci are intracellular, and though pyocyanase rapidly dissolves the free cocci it does not attack the leukocytes. The property of pyocyanase as a disinfectant of the pharynx was then tested. An epidemic of grippe in a children's ward was found to be due to Micrococcus catarrhalis, which was often and repeatedly grown in pure cultures from the nasopharynx of the patients. One or two applications of pyocyanase in 0.5 to 3 c.c. doses were sufficient to destroy the organism, and cultures made subsequently never contained Micrococcus catarrhalis. Further, Jehle made cultures from the nasopharynx of 155 persons who were either suffering from meningitis or were in contact with meningitic patients. Cultures from 39 of these individuals gave growths of meningococci; sometimes in a pure state. After one or two instillations of pyocyanase, meningococci disappeared entirely from the nasopharynx. Jehle thinks that pyocyanase will prove a valuable disinfectant for the nose and throat of individuals infected with these two organisms. It is not useful for all bacteria, however, for experiments with streptococci, staphylococci, pneumococci, and the colon and the Friedländer bacillus gave poor or negative results.

Experimental Studies upon Acute Toxic Nephritis.—It has always been difficult to decide what one should call degenerations of the kidney and what nephritis. Ribbert restricts the term nephritis to those inflammations which involve the vessels and are accompanied by exudation and proliferative changes, and Friedrich Müller takes much the same view, suggesting the term nephrosis for the degenerative changes which affect the epithelium of both the tubules and glomeruli. Cohnheim, on the other hand, recognizes a true tubular nephritis. Aufrecht distinguishes two main types of nephritis: a variety in which the epithelium of the tubules is affected, corresponding to the type of acute nephritis; and a variety in which the process is vascular and starts as a thickening of the walls of the vasa afferentia. Changes in the tubular epithelium occur later and the end result of this process is the "Schrumpfniere."

SCHLAYER and HEDINGER (*Deut. Arch. f. klin. Med.*, 1907, xl, 1) have made some interesting studies upon thes questions in rabbits poisoned by various substances. One of the important parts of the work was to measure the contraction and dilatation of the vessels by means of the Cohnheim-Ray oncometer. Variations in contraction and dilatation of the bloodvessels, and consequently the amount of blood in the kidney, were brought about in various ways. Under normal conditions after sensory stimuli the general blood pressure rose slightly and the vessels of the kidneys contracted; injections of adrenalin produced a greater rise in blood pressure and more marked contraction of the renal vessels. Injections of salt solutions and of caffeine did not give rise to any alteration of blood pressure, but caused an extreme grade of dilatation of the renal vessels which was accompanied by polyuria. Phloridzin produced glycosuria and polyuria without altering the blood pressure or the volume of the kidney.

In the first series of experiments nephritis was produced by poisoning the animals with chrome salts and with corrosive sublimate. These substances give rise to great destruction of the tubular epithelium without doing much damage to the glomeruli. The urine is plentiful, contains much albumin and enormous numbers of casts with detritus, but few blood cells. During the earlier stages of the intoxication the renal vessels, as measured by the oncometer, show normal or increased contraction following sensory stimuli and injections of adrenalin, and equally well-marked dilatation associated with polyuria and diuresis after injections of salt solution and caffeine. As the lesions in the kidney progress the vasodilatation and diuresis fail and after enormous destruction of the tubules with moderate changes in the glomeruli the vaso-dilatation and diuresis ceases almost completely though the power of vaso-constriction is well maintained. The reason for this gradual loss of vaso-dilatation was thought at first to be the mechanical hindrance of the dilatation of the vessels of the glomeruli from pressure brought to bear upon these structures by the casts in the tubules, but this was abandoned after it was found that during the early stages ligation of the ureter, a condition which would cause increased intratubular tension, did not interfere with the dilatation of the bloodvessels. The true explanation was found in the fact that the bloodvessels themselves were injured, proof of which was found in a second series of experiments. The nephritis in these experiments was brought about by the administration of cantharides, arsenic, and diphtheria toxin. With cantharides and arsenic the changes were the same. The anatomical lesions consisted principally in changes in the glomeruli. Moderate alterations were seen in the tubular epithelium, with casts in the tubules, but the glomeruli, especially in the severe cases, showed marked proliferation of the cells, with leukocytic infiltrations. The urine was scanty, contained relatively small amounts of albumin, great numbers of red blood cells, but only moderate number of casts. The changes in the contractility of the bloodvessels were entirely different from what was seen with the chrome salts and corrosive sublimate poisoning.

Even after small doses of cantharides and arsenic the power of dilatation of the vessels was almost entirely lost, and at the same time there was a disappearance of diuresis; with prolonged or more intense intoxication, total insufficiency of the renal vessels supervened and the elimination of the urine ceased almost entirely. These changes occurred with lesions in the kidney which did not approach in intensity the destruction following injections of sublimate and chrome salts.

Schlayer and Hedinger, from these experiments, distinguished two different types of acute nephritis: intoxication by chrome salts and corrosive sublimate causes a tubular nephritis, intoxication by cantharides and arsenic produces a vascular nephritis. Diphtheria toxin lies between the two, starting possibly as a tubular nephritis and ending certainly as a vascular nephritis. These experiments have, further, certain bearing upon the physiology of the kidney in general. It is evident that albumin can be eliminated from the glomeruli as well as from the tubular epithelium. The formation of casts has nothing to do with the bloodvessels, but takes place in the tubules. Every severe injury to the bloodvessels gives rise to a decrease in diuresis. With well-marked vessel changes phloridzin glycosuria no longer takes place. In extensive acute glomerular nephritis the function of the entire vascular system of the kidney is destroyed. Vascular nephritis is accompanied in its acute course by a low blood pressure, which may be due to the fact that the entire vascular system is injured by the poison producing the particular form of nephritis.

A Complex Hemolysin of the Pancreas.—FRIEDEMANN (*Deut. med. Woch.*, 1907, xxx, 585) finds that the fluid from a pancreatic fistula in animals contains a hemolysin which may be activated by lecithin, and which is comparable to the lecithins of the venoms of snakes and the poisons of bees. The pancreatic gland, moreover, contains a complex hemolysin which may be extracted with alcohol. This hemolytic substance may be activated by blood serum or by the alcoholic or ethereal extract of blood serum, but it cannot be activated by lecithin. It acts like an amboceptor, inasmuch as it is bound to the red blood corpuscles at a temperature of 0° C., but differs from an amboceptor in its high degree of thermolability. Small amounts of this pancreatic extract are capable of deviating the complement; and this power and the hemolytic power seem to be combined in one and the same substance. The substance is autohemolytic. Besides these properties the extract of the pancreas contains a toxin and a hemorrhagic poison which are similar in their action to crotalus toxin.

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ORIGINAL ARTICLES.

THE MEDICAL VS. THE SURGICAL TREATMENT OF GASTRIC ULCER.[1]

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The last word concerning the treatment of gastric ulcer has not and cannot be said, because, although individuals with large experience may give reasonably mature conclusions, the cases reported are meagre, particularly as to the after-history. The medical and the surgical reports are equally culpable. The latter may be excused because operative procedures have been instituted so recently that the after-history is not of value. Technique and operatve mortality have concerned the surgeon chiefly. Indeed, the former has so recently been fixed upon with any uniformity that the essential end results cannot be calculated. Fortunately, the time is approaching when definite conclusions can be drawn.

The difficulties attendant upon a solution of the best methods for the treatment of gastric ulcer are great. The uncertainty of its clinical course renders statistics concerning therapy most confusing and often unreliable. It is often present without symptoms; its most active manifestations may be followed by a period of quiescence; it may recur at varying intervals; it may remain a mild disease, large groups being attended by minimum mortality; or it may be a grave affection, vicious to the extreme.[2] There is, or may be, ever present, that general nervous state or nutritional fault which precedes, attends, and therefore invites recurrence of ulcer.

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[1] An address delivered at the Seventh Triennial Congress of American Physicians and Surgeons, Washington, D. C., May 7, 8, and 9, 1907.

[2] A physician of large experience in hospital and private practice, writes that he has never seen a death from gastric ulcer and never sent a patient to a surgeon. His experience of gastric ulcer was not large, it is fair to say.

In a general way the methods of treatment are either medical or surgical. At least it has been forced on the minds of the profession that gastric ulcer must be considered either a medical or a surgical disease. This attitude is such that, notwithstanding the protests of good surgeons, cases are forced upon them for operation when it would be unjustifiable to operate.

To combat this notion it seems necessary to assume that there are two forms of treatment; to demonstrate if possible their relative values; to show that each has a field and also in which field the respective methods apply. In one sense no comparison can be made. Gastric ulcer has two phases: one of simple course, the other with complications. These are the divisions held to in this paper—simple ulcer and ulcer with complications. The simple and the complicated phase have an entirely different clinical expression, although both are liable to the accidents of hemorrhage and perforation. The former, barring accidents, we hope to show, is a medical, the latter a surgical, affection.

Any form of successful treatment should lower the mortality of the disease, should aim to cure it so thoroughly that, as far as can be said, the ulcer is healed and that danger of relapse is not likely to occur, and that the patient is in as good condition relatively as before the onset of the affection. Any procedure which brings about such an ideal condition is of course the one to adopt. It is the purpose of the statistics which follow to show which form may more nearly attain this end. The problem is rendered difficult because the disease is not common and the experience of individual observers is small.

THE CRITERIA OF CURE. What constitutes a cure? The statistics of gastric ulcer show that most observers consider the disease cured when, after three or four weeks of treatment, the symptoms are relieved or removed. This is unfortunate. No value can be attached to cases reported in such manner. It is well known that at varying intervals after an alleged cure, perforation or hemorrhage may occur. To establish criteria on which to base a cure is difficult. It would seem that a lapse of two years without symptoms, that an approach at least to normal chemical conditions, and a removal of all abnormal physical or mechanical states are sufficient grounds to affirm that a cure has taken place in simple ulcer. To these some authorities would add the absence of occult blood from the stools for a long period. After a course of treatment by any means, notwithstanding these precautions, it is difficult to say that a cure has been obtained unless all the above conditions have been fulfilled.

THE ADVANTAGES OF MEDICAL TREATMENT. The two forms of treatment held out to us have advantages and disadvantages.

By the medical treatment the functional derangements, causal and secondary, are managed. It is conducted without doing violence to the body in any way, and if successful leaves the subject in as good condition as before.

THE DANGERS OF MEDICAL TREATMENT. A sense of false security is given, as the ulcer may be dormant and an accidental and fatal complication may arise when one is not prepared for it. The freedom from symptoms may flatter the patient that a cure has been established and induce carelessness in dietetics, hygiene, etc. Further, the healing of the ulcer, also possible under surgical treatment, may be attended by grave sequels of adhesions, contractions, or stenosis.

THE ADVANTAGES OF SURGICAL TREATMENT. By the surgical treatment, if indicated, the advantages of the medicinal treatment are secured. The operation is too often held to be the panacea, when success is really due to the after-cure. There is no reason why a patient who has undergone surgical treatment should not have an after-treatment like that which is given to cure the tendency to ulcer. The advantages depend upon the nature of the operation and the ability to deal with the complications. If the operation of excision is resorted to, the symptoms and accidents are prevented, providing, of course, there is only one ulcer. The gastro-enterostomy now in vogue is a procedure which while it relieves the many symptoms, does not warrant the belief that a cure is established. It is not proved that the ulcer has healed. Both perforation and hemorrhage occur after gastro-enterostomy. It is not known that relapse or recurrence is less frequent than in ulcers treated medically. The splendid statistics of Robson and of Mayo in their end-results compare favorably with, but are not better than, similar statistics of men who have treated similar groups of cases medically. The time element in the surgical cases is the unsettled factor. If the ulcer is excised, a dangerous and not practical procedure we are told, or destroyed by perforation, the only risk comes from persistence of the organic cause or the presence of two or more ulcers. In the second period of ulceration, when complications have arisen, the advantages are entirely in favor of surgery.

THE DANGERS OF SURGICAL TREATMENT. (1) The same as in the medical treatment unless excision of the ulcer, if solitary, is resorted to. (2) Operative death. (3) Death from complications. (4) Death or ill health from defects in metabolism. (5) The effect of the operation. The patient on whom an operation has been performed has the disadvantage of a psychic insult, the influence of which is not trifling, and of being in possession of new physical and physiological conditions the nature of which time alone can tell. It must be admitted that a patient who has undergone a grave surgical operation is not in as good condition to have that indefinable something, which may be termed neurosis, cured, as he who has not been submitted to such an insult.

The surgical dangers are constantly lessening. From an average operative mortality of 20 per cent. they have fallen to 1.5 to 2 per cent. for individual operators, and 10 per cent. for all. The tables will show a remarkable change born of experience in technique between the operative results of all prominent operators in their first and their latest series. Thus the vicious circle is rarely established and in the latest large group of cases of the Mayos, Moynihan, Robson, Deaver, and others it did not occur. Peptic jejunal ulcer is also a less frequent complication. (See Lyle and Joslin.)

Hunsheimer, Joslin, Rosenberg, Kreuger, and others have studied the effects of gastro-enterostomy on metabolism and on gastric secretion. The studies are not sufficient as to the metabolism, although most suggestive. More studies must be made also of the chemical secretions. Kreuzer's[3] are the most recent.

The after-effects of gastro-enterostomy are, he states: (1) Secretory function again assumes the normal condition. (2) Total acidity falls to the normal—sometimes below, but rises shortly to the normal. If the acidity was above normal before operation it sinks otherwise to normal. If acidity was diminished the usual rule is for it to reach normal or slightly above. (3) Free hydrochloric acid—usually lessened after operation. (4) In those cases in which no trace of hydrochloric acid is found, it is obtainable after the operation, the exception being to find it missing. A deficient hydrochloric acid cannot be considered a disadvantage, as it is only a result of increased emptying power of the stomach. (5) The backflow of bile into the stomach disappears gradually and does not seem to cause any pronounced symptoms. (6) Backflow of pancreatic juice is seldom recognizable. Complete blocking of the outflow seems to be more the result of the disease than of the operation.

Katzenstein studied the question experimentally and compels us to assume that the secretory, in addition to the motor, function is benefited by operation. Not only did he find that the backflow of bile and pancreatic juice lowered the acidity because of this alkalinity, but also that the delicate reflexes of the duodenum and jejunum were not lost, if the animal was fed on special foods, as fats.

The experimental evidence does not forbid surgical procedures, apparently.

A STUDY OF STATISTICS. To one who has had the opportunity of studying only some sixty odd cases of gastric ulcer, the wisdom that comes from personal experience cannot be overwhelming. The study here presented is based, therefore, on an analysis of 1871

[3] Effects of Gastro-enterostomy on the Chemical Functions of the Stomach, 1906.

cases of gastric ulcer and 316 cases of duodenal ulcer collected from the accumulated literature since 1897.[4] The cases selected are those only which have been reported in full detail.

The German and French cases[5] were tabulated in accordance with the following scheme: (1) Age. (2) Sex. (3) Hemorrhage. (4) Pain. (5) Vomiting. (6) Boas' tender points. (7) Acidity. (8) Feces. (9) Retention. (10) Duration of diseases. (11) Medical treatment. (12) Result. (13) Surgical treatment. (14) Result. (15) Length of period under observation after treatment. (16) Recurrence. (17) Remarks. (18) Observer.

The English and American cases, on the other hand, have been analyzed to determine the incidence of age and sex; the basis for diagnosis according to symptoms and gastric analyses; the occurrence of retention from simple dilatation, pyloric spasm, adhesions, hour-glass contraction, or pyloric obstruction; the duration of the disease; the medical treatment, if any, and the result; the surgical treatment, if any, and the result; the duration of relief; and recurrence. The question of hyperacidity or hyperchlorhydria was, through an oversight, omitted. This is to be regretted, as the division of ulcers into those with secretory symptoms and those with motor symptoms should be clearly defined. Fortunately the answers to the various questions are such that in a general way this question has been satisfactorily met.

In addition a study of 586 cases has been made, based upon private communications received in reply to a letter to members of the Association of American Physicians which requested an anwser which would supply the above facts. I scarcely need say I am deeply indebted for the valuable contributions thus placed at my disposal.[6]

I have kept apart from my tables and used as control, for analysis and comparison, the unique and unparalleled experience of the Mayos and of Robson, Moynihan, Munro, v. Mikulicz, and many others, as well as of the distinguished gentlemen who took part in the discussions of the Royal Medical and Chirurgical Society in 1906. No papers on gastric ulcer can be written without drawing fully on the older writings and the not remote ones of Welch, Fenwick, Lebert, Dreschfeld, Brinton, and others.

To control the errors liable to occur in statistics, comparisons have also been made with the conclusions of the various able writers who have analyzed large groups of cases. The skilful analyses of Wier and Ford, Rodman, Patterson, Caird, Russell, Bramwell, Greenough and Joslin; of Lund, Joslin and Murphy; of Sears,

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[4] The cases of duodenal ulcer have been reserved for a future communication.

[5] Over 900 cures. Treatment only is analyzed in this communication.

[6] Since this has been written, valuable private communications with lists of cases have been sent me by Deaver and others. They will be embodied in an analysis to be published in the future.

Campbell Howard, Thompson, and others are invaluable. The German and French theses and the papers of Brunner, Kreuzer, Krönlein, Körte, v. Mikulicz, Minkowski, Blumensath, Spicker, Schneider, Becker, Ladevèze, and Rochard have also been excellent controls.

The well-founded objection that statistics which give the results of treatment are usually exaggerated, whether surgical or medical, because the tendency to omit failures is dominant, has been borne in mind. It does not appear, however, that there is any way of bringing together a large number of cases except by this means.

The statistics offered by the advocates of medical treatment do not fully prove their case. A better brief could have been made by them had the cases been reported in greater detail and especially, also, with more precise accounts of after-results. It must be said in this respect that the surgeons have their cases better in hand for continued observation. This has grown out of the necessity before them of continually proving the contention that their invasion of the sphere of medicine is warranted by results. The quarrel as to the respective merits of the medical and the surgical treatment of appendicitis would be in continuance had the surgeons not been able to marshal their facts to telling purpose. Then, too, the responsibility of taking the life of the patient in their hands adds to the necessity of scientific precision of knowledge instead of glittering generalities.

The study of statistics impresses one with the value of having a single guiding head and hand for each department of a hospital and of a liberal management which will employ such aid as can enable the staff properly to collect records and to keep in touch with patients after their discharge. An efficient medical secretary is as essential as a good nurse in any institution. Only by these means can fruitful observation be secured. Moreover, although it places another burden on the practitioner, if he wishes to attain knowledge that can be applied to his daily work, he must similarly keep in touch with patients after their discharge.

As previously intimated statistics are thus far not reliable, and can only admit of approximate conclusions because no established criteria have been formed for a basis of calculation. They are meagre because the patients are lost sight of. From the studies of the many cases in the literature I should say we need information of the end-results and the course of the disease during a period of ten years. We should agree upon what constitutes a "cure." All realize the difficulties, whether it is to base the result on the duration of relief of symptoms and the absence of physical sequels, or on the plan of Boas, to affirm a cure only when occult blood (in addition to the above) is absent from the stools.

The study has not been satisfactory, for the reasons above mentioned. It has been profitable because it has laid bare our deficiencies. Many points of interest, however, are brought out, and the province of the physician and the surgeon defined.

Some elementary facts obtained from a study of the statistics of gastric ulcer must be known in order to estimate the relative merits of remedial measures. We should have a clear notion of the frequency of gastric ulcer, of the mortality of the disease irrespective of treatment, and of the mortality following the surgical and the medical treatment of the affection.

THE FREQUENCY OF GASTRIC ULCER. The infrequency of gastric ulcer, as the following statistics will show, behooves us to pause before urging operative procedures for intractable indigestion, hematemesis, gastric pain, and other isolated symptoms on the ground that they are of ulcer origin. I am willing to admit that an exploratory operation in competent hands is attended by very little danger. If the surgeon wishes to coddle his mental powers by employing this means of diagnosis and the patient is willing to submit, nothing more need be said. Otherwise, gastric ulcer will not be considered a frequent surgical affection. This is surely the case in simple ulcer. It would be infrequent in complicated ulcer, if the simple ulcer were treated for a sufficiently long period of time.

FREQUENCY OF GASTRIC ULCER BASED ON AUTOPSIES.

Author.	Number of autopsies.	Number of gastric ulcers.	Per cent.
Welch (German statistics)	32,052	1522	5.00
Dietrich	10,103	350	3.40
Fenwick	47,912	2019	4.20
Harlow Brooks (Bellevue Hospital)	1,000	9	0.90
Pearce (Albany Hospital)	1,000	11	1.10
Welch (Bellevue Hospital)	800	6	0.75
Willigde	3,400	221	0.64
Howard (Hospitals of United States)	10,841	144	1.32
Francine (Philadelphia and Phipps)	2,937	41 (duod. & gastric)	0.13
German Hospital (A. O. J. Kelly)	937 (11 years)	13	0.13
Mallory (Boston City Hospital)	2,600	25	0.90
Warthin	200 1895–1899	7	0.35
Schneider (Munich)	4,535	89	1.92

Howard, in a valuable paper pointed out that the frequency of ulcer varies in different cities. Based on clinical returns he found the largest percentage of cases in Boston—1.28; the smallest number in Denver—0.12. The Boston statistics tallied well with the figures of Greenough and Joslin from the Massachusetts General Hospital, where a percentage of 1.74 was found. It is of interest to note that in Breslau and Zurich 0.66 per cent., London 0.78 per cent., Berlin 1.33 per cent., Edinburgh 2.2 per cent., are fair estimates of the clinical frequency of gastric ulcer. The percentage of frequency in America is greater than that in Germany and London. Edinburgh is higher than America or London.

See Correction on Opposite page.

FREQUENCY OF GASTRIC ULCER BASED ON CLINICAL OBSERVATION.

Author.	Number of cases.	Number of gastric cases.	Number of cases of ulcers.	Private practice.	Per cent.
Lebert	41,688	...	252	...	0.6400
Charité Hospital, Berlin, 1888-1898	42,219	...	555	...	1.3300
Howard*	(Med. adm.) 161,599	...	930	...	0.5700
Fenwick †	(Med adm.), 45,712 Out-patients, 5,000	... ...	383 37	... ...	0.8200 0.7400
Bramwell ‡	In-patients, 35,692 Out-patients, 7,665	... ...	803 76	... ...	2.2000 0.0900
Medical service of Bellevue Hospital, Harlow Brooks	In 3 months, 6,205	...	3	...	0.0040
Berlin Charité	1903-04, 15,834 1904-05, 15,863	250 279	46 48	... ...	0.0022 0.0030
Clayton §	1,869	108	8	...	0.0040
Elsner	1,000	171	14	Private.	1.5000
Ingers	2,105	550	7	Private.	0.0300
Lichty	6,870	1,269	103	Private.	1.4000
Friedenwald	13,794	...	287	Private.	2.0000
Sawyer	....	1,800	63	Private.	

* Hospitals of United States (15). † London and Temperance Hospitals. ‡ Edinburgh Royal Infirmary. § Garfield Hospital.

The variation in statistics in hospitals of the same city is due to the different class of patients admitted to the respective hospitals. It is well known that the death rate and frequency are higher in Guy's Hospital in London than in other hospitals, because of the poor class of patients admitted to the former.

In private communications received from physicians throughout the country the reports of the infrequency of gastric ulcer in the cities mentioned above is confirmed. It must not be forgotten, however, that different observers have different criteria upon which the diagnosis of ulcer is based. Thus Howard states that the disease is rare in Baltimore, whereas Friedenwald reports an unusually high percentage. Professor Baumgarten in St. Louis writes that it is very infrequent and that he has never had to send a case to a surgeon.

THE MORTALITY. The mortality of gastric ulcer varies with different observers, as the tables indicate.[7] Irrespective of the

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[7] With a view to economy of space, many of the tables are omitted. Those especially interested in them will find them in detail in the Trans. Cong. Amer. Phys. and Surgeons, 1907, p. 56.

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SUPPLEMENT TO

THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES

MAY, 1908

Dr. John H. Musser regrets that inadvertently certain errors were made in computing the percentages in several of the tables that accompany his article on the "Medical *versus* the Surgical Treatment of Gastric Ulcer," published in the AMERICAN JOURNAL OF THE MEDICAL SCIENCES, December, 1907, vol. cxxxiv, page 781, and he asks the readers of the JOURNAL to take note of the corrections made in the subjoined tables:

FREQUENCY OF GASTRIC ULCER BASED ON CLINICAL OBSERVATION (page 788).

Author.	Number of cases.	Number of gastric cases.	Number of cases of ulcers.	Private practice.	Per cent.
Lebert	11,688	...	252	...	0.60
Charité Hospital, Berlin, 1888–1898	12,219	...	555	...	1.31
Howard[1]	(Med. adm.) 161.599	...	930	...	0.57
Fenwick[2]	(Med. adm.), 45,712	...	383	...	0.84
	Out-patients, 5,000	...	37	...	0.74
Bramwell[3]	In-patients, 35,692	...	803	...	2.25
	Out-patients, 7,665	...	76	...	0.99
Medical service of Bellevue Hospital, Harlow Brooks	In 3 months, 6,205	...	3	...	0.01
Berlin Charité	1903–04, 15,834	250	16	...	0.29
	1904–05, 15,863	279	48	...	0.30
Clayton[4]	1,869	108	8	...	0.43
Elsner	1,000	171	14	Private	1.40
Ingers	2,105	550	7	Private	0.33
Lichty	6,870	1,269	103	Private	1.49
Friedenwald	13,794	......	287	Private	2.08
Sawyer	....	1,800	63	Private	

[1] Hospitals of U. S. (15). [2] London and Temperance Hospitals. [3] Edinburgh Royal Infirmary. [4] Garfield Hospital.

FREQUENCY OF GASTRIC ULCER BASED ON AUTOPSIES (page 787).

Author.	Number of autopsies.	Number of gastric ulcers.	Per cent
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Dietrich .	10,103	350	3.46
Fenwick . .	47,912	2019	4.21
Harlow Brooks (Bellevue Hospital)	1,000	9	0.90
Pearce (Albany Hospital)	1,000	11	1.10
Welch (Bellevue Hospital)	800	6	0.75
Willigde . .	3,440	221	6.42
Howard (Hospital of U. S.)	10,811	144	1.33
Francine (Philadelphia and Phipps)	2,937	41 (duod. & gastric)	1.39
German Hospital (A. O. J. Kelly)	937 (11 years)	13	1.38
Mallory (Boston City Hospital)	2,600	25	0.96
Warthin . . .	200	7	3.50
Schneider (Munich)	1895–1899 4,535	89	1.96

PERCENTAGES OF SIMPLE ULCER, SEQUELS, HEMORRHAGE, PERFORATION IN CASES COLLECTED FROM THE LITERATURE (page 789).

Total number of cases 1871:

Number of cases perforation	536	28.6 per cent
Number of cases hemorrhage[1]	160	8.6 "
Number of cases simple[2]	409	21.9 "
Number of cases sequels	766	40.9 "
	1871	100.0

[1] Demanding treatment

[2] Without serious hemorrhage or sequels

method of treatment the mortality of patients admitted to the hospital seems to be from 6 to 8 per cent., although Bulstrode places it as high as 18 per cent., Gilman Thompson at 20.5 per cent., and Körte and Herzfeld at 25.9 per cent. In private practice the mortality is much less, as the cases of Friedenwald, Lichty, and others show.

Percentages of Simple Ulcer, Sequels, Hemorrhage, and Perforation in Cases Collected from Literature. Total number of cases 1871: Simple ulcer[8] 409 (21 per cent.); perforation, 536 (28.1 per cent.); hemorrhage,[9] 160 (8.01 per cent.); of sequels 766 (40.01 per cent.). The number of reported cases of simple ulcer included in the analysis is undoubtedly very small. As no special interest is attached to single cases treated medically, they are not reported. Moreover, the proportion of cases is not true. Thus, cases of perforation have been reported more frequently of late, to clear up questions of diagnosis and settle modes of surgical technique. The unpublished cases from private practice give a truer idea of the relations of the simple and the complicated ulcer and the frequency of the grave accidents in various forms:

Percentages of Simple Ulcer, Sequels, Hemorrhage, and Perforation in Unpublished Cases. Total number of cases, 586. Simple ulcer, 194 (33.1 per cent.); hemorrhage (grave), 23 (3.9 per cent.); perforation, 16 (2.7 per cent.); sequels, 353 (60.2 per cent.).

Simple Ulcer. The results of the treatment of simple ulcer in cases collected from the literature is shown in the following table:

Simple uncomplicated ulcer.	409 cases				21.0 per cent.	
			Cure and improvement		76.0 "	
Male	143	34.9 p. c.	Unimproved		6.6 "	
Female	266	65.1 "	Death		17.3 "	
Immediate result.	Surgical treatment,		Cure	184	68.1 p. c.	73.2 p. c.
			Improved	41	5.1 "	
			Unimproved	18	6.6 "	26.6 p. c.
			Death	54	20.0 "	
Immediate result.	Medical,		Cure	102	73.3 "	81.2 p. c.
			Improved	11	7.9 "	
			Unimproved	9	6.4 "	18.4 p. c.
			Death	17	12.4 "	

The mortality is 17.3 per cent. It is seen that the advantage is in favor of medical treatment, as the immediate result from such treatment is attended by a mortality of 12.4 per cent., while by surgical treatment it is 20 per cent. We must admit, physician and surgeon alike, that simple uncomplicated ulcer is a medical disease.

This table does not include the statistics of Leube, Lichty, and many others. Hemorrhage and perforation cases are not included.

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[8] Without serious hemorrhage or sequels.

[9] Demanding treatment

Thus, Leube and Friedenwald have, and Bramwell, Schultz and Robinson estimate, a mortality below 6 per cent. Without doubt the excellent results attained in private practice are explained by the fact that the patients apply early for treatment, at once are placed on a proper dietary, and medical supervision is continued for a long time. It must be remembered that hospital patients, presumably the poorer classes, have more frequent complications and sequels and a higher mortality than private patients. This occurs because the former class cannot carry out treatment for a sufficiently long time, and, indeed, usually give it up as soon as symptoms are relieved.

The results of the treatment of simple ulcer in private practice are shown in the following table (unpublished cases):

Simple ulcer.	Total number of cases	194			
	Male	56	28.9 per cent.		
	Female	138	71.1 “		
35 Surgical treatment	18.0 per ct.	Cure	85.70 per ct.	Unimp.	14.3 per ct.
159 Medical treatment	82.0 “	Cure	91.70 “	Unimp.	08.3 “
As to decade of surgical treatment, most cases between 20-30.					
	48.5 per ct.	Men	29.49 per ct.	Women	70.6 per ct.
Of these cure	94.1 “	Men	29.40 “	Women	70.6 “
unimp.	05.9 “	Women	100.00 “ (one case).		
As to decade of medical treatment most cases between 20–30.					
	48.4 per ct.	Men	20.50 per ct.	Women	79.5 per ct.
Of these cure	91.0 “	Men	17.90 “	Women	82.1 “
unimp.	09.0 “	Men	28.50 “	Women	71.5 “

A closer analysis shows that of the 85.7 per cent. that were noted as cured by surgical treatment, 77.1 per cent. were reported as cured finally and 8.5 per cent. improved. Of the 14.3 per cent. unimproved, 11.4 per cent. died. 91.7 per cent. were reported as cured by medical means, of which 60.1 per cent. were cured absolutely and 32 per cent. improved. 5 per cent. of the unimproved remained ill and 3.1 per cent. died.

The death rate in simple uncomplicated ulcer treated surgically is higher (11.4 per cent.) than the death rate of cases treated medically (3.1 per cent.). It must be observed, however, that a larger number were reported "improved," after medical treatment, the end of which is not known, than after surgical treatment.

A study of the 270 cases of simple ulcer treated surgically shows that the patients came to the surgeon after the thirtieth year and the larger proportion in the decade from 40 to 50. Male subjects deferred treatment to a later period than females.

Cases are treated medically in the earlier decades. They are in all probability uncomplicated. The surgeon is brought to the complicated cases. Either one of two things is evident: cases are not sufficiently grave to undergo treatment, or medical treatment is not conducted as vigorously and over as long a period of time as is required to bring about a cure. It is more likely that the early symptoms were absent, mild, or overlooked and treatment was not needed until organic conditions developed. In private cases the surgical treatment was instituted with the same relative degree of frequency as the medical in the corresponding decades. The tables show that the reported or hospital patients (poorer classes) delay treatment and that the more severe cases go to the surgeon. In private practice there is not the same delay.

In simple ulcer the death rate under surgical treatment was 20 per cent., and under medical treatment 12.4 per cent. Privately reported cases showed a surgical mortality of 11.4 per cent. and a medical mortality of 3.1 per cent. It is not easy to compare these statistics with others, for in other analyses the mortality is given of both simple and complicated ulcer when not due to hemorrhage or perforation. The results of the medical treatment of peptic ulcer in the Boston City Hospital as reported by Sears showed a mortality of 21 per cent. Greenough and Joslin in a study of 187 cases, treated in the Massachusetts General Hospital, found the mortality was 8 per cent. It must be noted that only 40 per cent. of the 82 per cent. discharged cured or relieved remained well. The series which Shultz analyzed from the Breslau and New Hamburg Hospital gave a mortality of 5.4 per cent., or if unrelieved and died are added, 10.5 per cent. unimproved by treatment. Sears well points out that the longer cases are followed the higher the proportion of failures, rising at the end of five years to 50 per cent. Unfortunately neither surgical nor medical cases have been observed over the length of period that permits of definite conclusions. Gradually there comes to light more and more cases in which repeated operations were done, indicating failure by surgical as well as by medical means. Hewes reports a series of cases of simple ulcer which more nearly correspond to our groups. Of 51 cases immediate results with complete relief followed medical treatment in 48; failure in relief of symptoms, 2; died, 1. 49 of the 51 were followed for a period of two years with results as follows: 31, or 63 per cent., cured; 18, or 36 per cent., recurred; 1, or 2 per cent., died. The same class of cases treated surgically, 13 in number, resulted as follows: cured 6, or 46 per cent.; recurred or not relieved by operation 3, or 23 per cent.; died shortly after operation 4, or 30 per cent. A separation of the cases into the simple and complicated cases was not made in B. Robinson's cases (mortality 2.1 per cent.); in Bramwell's (mortality 6.2 per cent.); in Russel's (mortality 2.1 per cent.); and von Leube's (mortality 2.4 per cent.); all of which were treated medically. Had such been the case the mortality for the management of simple ulcer would have been less without doubt.

Remote Results of the Surgical and the Medical Treatment of Simple Ulcer (Literature).

	Simple Ulcer, Medical.				*Simple Ulcer, Surgical.*			
Period	Cure.	Imp.	Unimp.	Death.	Cure.	Imp.	Unimp.	Death.
6 months	20	10	2	11	36	5	4	47
6-12 months	3	0	0	0	28	1	0	0
1-2 years	0	0	0	0	43	0	0	0
2-3 years	0	0	0	0	6	0	0	0
3-4 years	1	0	0	0	12	1	0	0
4-5 years	0	0	0	0	1	1	0	0
5 years	1	0	0	0	4	0	0	0
Not given	85	..	5	..	..	109	4	.

This table shows the result from six months to five years after treatment. It shows also the unsatisfactory state of the literature, inasmuch as end results are disregarded by surgeons and clinicians alike. Nevertheless, of 270 cases treated surgically, presumably cured, 47 were dead at the end of six months, while of the 139 treated medically 11 died in the first six months.

Remote Results of the Medical and the Surgical Treatment of Simple Ulcer (Unpublished Cases).

	Medical.				*Surgical.*			
Period.	Cured.	Imp.	Unimp.	Death.	Cured.	Imp.	Unimp.	Death.
1-6 months	6	6	1	5	2	..	..	4
6-12 months	9	3	..	..	2	..	..	..
1-2 years	4	2	..	..	..	..	..	..
2-3 years	7	..	..	..	..	..	..	..
3-4 years	..	1	..	..	..	..	..	..
4-5 years	..	..	..	..	..	..	..	..
Over 5 years	1	..	..	..	..	..	..	..
Not given	58	36	6	..	18		..	..

Recurrence noted in 2 cases.

In addition to the foregoing the records show 35 cases apparently cured but followed by recurrences. Of these 29 showed benefit after further treatment. Of the cases treated medically recurrence was noted in 16 apparent cures. Although the period of observation was not given, all but 14 were observed for periods extending beyond six months.

The remote results of the treatment in simple gastric ulcer are determined with difficulty, inasmuch as most of the cases analyzed again are not divided into simple and complicated. Mayo Robson had followed patients for from one to five years and was able to find 96 out of 112 who had suffered from gastric ulcer, for whom he had operated, in good health. Unfortunately his cases are not classified as to sequels. The same may be said of the studies of Greenough and Joslin, Russel, Debove, Murdock, and others. It is proper to note that many insist that the remote results of ulcer (with and without complications) treated medically are most unsatisfactory—56 per cent. of the 55 per cent. who were discharged from the Massachusetts General Hospital (Greenough and Joslin) were dead or suffered from recurrence after the lapse of five years. Five hundred patients suffering from gastric ulcer were admitted to the London Hospital, 42 per cent. of whom had previous attacks. Bulstrode, who made the analyses, states that 82 per cent. were discharged, relieved or cured, but concludes, if the large percentage noted above had recurrences, similar recurrences must have occurred in the 82 per cent. It is Mayo Robson's conviction that 25 per cent. of all cases of ulcer of the stomach treated medically succumb to the disease or its complications. Unfortunately, again we are without the divisions of the simple and the complicated ulcer. Debove and Remond estimate the mortality at 50 per cent.

In this connection the careful analysis of Russel of the results of the treatment of 47 patients (no division) should be given: after a lapse of from two to thirteen years, the direct mortality amounted to 2.1 per cent.; 42.6 per cent. ended in recovery; 4.3 per cent. died from intercurrent disease; 6.4 per cent. could not be classified; and 44.7 per cent. were suffering from stomach symptoms. Hawkins, in a study of 536 cases, admitted to St. Thomas' Hospital, found the mortality 13.3 per cent., of which 0.9 per cent. died of hemorrhage, 8.5 per cent. of general, and 1.9 per cent. of local peritonitis. Adhesions and pyloric stenosis caused the death of 1.9 per cent. Patterson reports 99 cases cured; 9 result fairly good, and 8 unsatisfactory (4 died and 4 relapse) in 116 gastro-enterostomies, after a lapse of eighteen months to nineteen years. Again there is no statement of the sequels, as in the following list treated medically, which in 72 cases resulted in 19 cures, 2 probable cures, 5 probably not cured, and 46 not cured. Only 7 of the 19 remained free from symptoms. Von Leube's 556 cases gave a mortality of 2.4 per cent.; cure, 74.1 per cent.; improved, 21.9 per cent., and unimproved 1.6 per cent. Huberman reports recurrence in 20 per cent. of 100 cases cured by ordinary medical treatment, and 8 per cent. of recurrence when the Lenhartz treatment was employed in 135 cases.

The opinion of most writers is that the medical treatment of simple gastric ulcer is attended by the best results, whether the outcome immediate or remote is considered, and the quoted statistics appear to uphold this contention.

THE TREATMENT OF HEMORRHAGE. The subjoined tables are an analysis of the cases which were treated, because life was in peril, specifically for hemorrhage. Our statistics support the observations of those who admit that hemorrhage is best treated medically, but under many circumstances an operation is justifiable. Hemorrhage occurred in 160 of the 1871 cases collected from the literature.

Majority of cases between 20 30, women.		Per cent.
I. 135 acute cases		53.2
77 were treated surgically		57.1
of these cure	41	53.2
death	33	42.8
not given	3	4.0
58 were treated medically		42.9
of these cure	36	62.0
improved	6	10.3
unimproved	0	
death	13	22.4
not given	3	5.3
II. 25 chronic cases		15.7
18 were treated surgically		72.0
of these cure	14	77.6
improved	0	
unimproved	0	
death	4	22.4
7 were treated medically		28.0
of these cure	1	14.3
improved		
unimproved		
death	2	28.5
Result not given	3	42.8

One case treated and died.

Hemorrhage occurred in 23 of the 586 (3.9 per cent.) remote cases.

I. Acute	20	86.9
Surgical cases	7	35.0
of these cure	4	42.8
improved	1	14.3
death	3	42.8
Medical cases	13	65.0
of these cure	10	76.9
improved	0	
unimproved	0	
death	3	23.1
II. Chronic	3	13.1
Medical cures	2	
Surgical cures	1	

The occurrence of hemorrhage in the course of gastric ulcer is placed at figures ranging from 50 per cent. to 80 per cent. of all cases. The figures include those which are small in amount, infrequent or single, and those of very frequent occurrence, multiple, and in which large amounts of blood are lost. Treatment is not necessary in many cases. In the other cases medical measures are resorted to. When life is in peril because of frequent small bleedings or because of one or more large hemorrhages putting life in peril, surgical measures have recently been considered. It is the consensus of opinion that hemorrhage is usually controlled by well-established lines of procedures. Can the few be saved by persistence in medical measures or by resort to surgery? Our statistics uphold the contention that acute large hemorrhage treated by medical means is attended by less mortality than that which is treated by surgical means; the percentage of death after medical treatment is 22.4, and after surgical treatment, 42.8. Of course, the cases have been those in which life was in peril, desperate from the beginning, and perhaps an unfair class for the surgeon. It is presumed that the question with each surgeon was one of operation in the face of death. In the unpublished cases the percentage of hemorrhage, of some types, was small and hence there are only a few upon which to base a calculation. The mortality in the respective divisions is about that given above—medical, 23.1 per cent.; surgical treatment, 42.8 per cent.

The mortality of hemorrhage when medical means are employed is given as follows: Becker, 17.2 per cent.; Brinton, Gerhard, and Welsh, 3.5 per cent.; Diller, 10 per cent.; v. Leube and Ewald, 1.125 per cent.

The remarkable experience of Moynihan stands out as an encouraging feature. This surgeon saved 19 out of 22 patients. Connell, in a paper published in 1905, finds a lessening of mortality, as follows: Marion (1897), 66 per cent.; Hartmann, (1898), 51.6 per cent.; Bidwell (1899), 50 per cent.; Robson (1900), 53.8 per cent. Rodman's analysis showed a mortality of 40 per cent., and Connell, in an analysis of 100 cases, found a reduction of the mortality in 57 acute cases to 26.3 per cent.

Chronic bleeding ulcer, leading to grave anemia, is cured by surgical measures, according to our analysis, more frequently than by medical means. The mortality surgically is 22.4 per cent.; medically, 28.5 per cent. In private practice, the cases are too few for analysis. The mortality of Robson's cases (1900) is 10.5 per cent.; of these analyzed by Rodman (1900), 19.3 per cent., and by Connell (1904), 8.5 per cent.

An analysis of the two classes of cases as to age and sex did not, as the tables show, reveal that either of these states predisposed to success or failure in any line of treatment. Greenough and Joslin note a mortality of 17 per cent. in males and 1.2 per cent. in females.

The individual operator stands out. The person of experience tallies higher than the group of operators of small experience. The conclusions seem warranted that the patient is in safer hands by medical treatment, unless he can have the services of a surgeon of large experience. Even then, in acute hemorrhage, unless life is despaired of, the surgeon prefers to postpone operation until the hemorrhage is controlled and the patient relieved of shock and acute anemia.

TREATMENT OF PERFORATION. The treatment of perforation has been discussed very exhaustively during the last ten years. The improved abdominal technique has led to remarkable successes in this field.

INCIDENTS OF PERFORATION (LITERATURE).

Total No. of cases, 1871.

Perforation 536 (28.1 per cent.).

Sex	
Male	191
Female	334
Not given	11

Age	
1-10	3
10-20	95
20-30	217
30-40	90
40-50	51
50-60	29
60-70	10
Over 70	5
Age not stated	36

Operations for perforation	481
Not operated	55
	536

INTERVAL BETWEEN PERFORATION AND OPERATION.

Number of hours.

	1-12	12-24	24-46	36-48	over 48	1 wk.	1-2 w.	2-3 w.	3-4 w.	over 4	Not given, acute. 58	Not given, chronic.
*C	144	73	21	8	3	12	4	1	...	17	32	1
D	38	43	12	17	19	4	0	1	...	4	26	1
	182	116	33	25	22	16	4	2		21	No op.	2
	Acute.					Subacute and chronic.						

* C, cured; D, died; I, improved; U, unimproved.

TREATMENT OF PERFORATION. LITERATURE. 536 CASES. In 481 operations—89 per cent. Cured 65.7 per cent. Died 34.3 per cent. In 55 without operation—Cured, 11 (20 per cent.); died, 30 (54.5 per cent.); not stated, 14. Sex: Male, 191 (35.6 per cent.); female, 334 (62.3 per cent.); not given, 11 (2.1 per cent.).

The majority of perforations occurred in females between the ages of twenty to thirty—201 cases: 142 women; 59 men.

The best percentage of cures was noted when operation was between one to twelve hours after perforation.

In cases of third decade—Cure, 135 (67.1 per cent.); women, 92 (68.1 per cent.); men, 44 (31.9 per cent.). Unimproved, 66 (32.9 per cent.); women, 51 (77.2 per cent.); men, 15 (22.9 per cent.).

In the same decade the medical treatment was as follows: 14 cases—11 women, 3 men; of these—cured 4 (28.5 per cent.), all women. Unimproved, 10 (71.5 per cent.); women, 7 (70 per cent.); men, 3 (30 per cent.).

PERFORATION. *Cases Collected from the Literature.* Period of observation after recovery from operation for perforation during which time patients remained well: one to six months, 93; six to twelve months, 38; one to two years, 13; two to three years, 14; three to four years, 3; four to five years, 3; five to ten years, 5; not stated, 131. Total number of cases of perforation, 536; total number of permanent benefit ranging up to ten years, 300.

Recurrence. After six months, none; after one year, 2; after two years, 1; after three years, none; after four years, 1; after five years, none; not stated, 1. Benefit, but later recurrence, 5.

Unpublished Cases. Number of cases, 16 (2.7 per cent.). Sex: male, 7 (42.7 per cent.); female, 9 (56.3 per cent.). Age: ten to twenty, 1; twenty to thirty, 4; thirty to forty, 6; forty to fifty, 3; fifty to sixty, 1. Time between perforation and operation not stated. Cured, 5 (31.2 per cent.); died, 11 (68.8 per cent.).

Perforation spares neither age nor sex, occurring in about the same degree of frequency that ulcer occurs in each sex and in the respective decades. Apparently it is a slightly more frequent accident in males. Contrary to the experience in hemorrhage, age is an influence which counts for or against special lines of treatment. It would be expected that in the extremes of life the mortality would be higher, with or without operation, than in the middle periods. Sex does not appear to have such influence.

The mortality of perforation as given by four observers is about as follows: With operation: Moynihan, 33.3 per cent.; Hawkins, 43 per cent.; Brunner, 48 per cent.; Garre, 30 per cent. Without operation: Brunner, 95 per cent.

Operation was resorted to in 481 of the 536 cases with a mortality of 34.3 per cent.; cures, 65.7 per cent. 55 cases were not operated on, death taking place in 54.5 per cent. of the cases. 20 per cent. of the 55 were cured and the result not given in the remainder of the percentage. In the unpublished or private cases 31.2 per cent. were cured by operation, and 68.8 per cent. died.

Great interest is attached to the time interval between the accident and the operation. The analysis shows, in consonance with that of others, that the shorter the interval the higher the percentage of cures. Weir and Foote and afterward Keen[10] analyzed reported cases, and Robson and Monihan and Caird presented individual experience. The figures below give the percentage of death:

	Weir and Foote.	Keene.
	Per cent.	Per cent.
Under 12 hours	39.13	19.23
12 to 24 hours	63.63	50.80
24 to 48 hours	77.77	55.55
Over 48 hours	57.51	25.31

	Caird.	Robson and Moynihan.
	Per cent.	Per cent.
Under 12 hours	5 cases, 40	49 cases, 28.5
12 to 24 hours	8 cases, 37.5	35 cases, 63.6
24 to 36 hours	3 cases, 33.3	14 cases, 87.5
36 to 50 hours	2 cases, 50	2 cases, 100.0

Goldsincker, in a summary of 250 articles, finds a mortality of 50 per cent., and in 236 cases 51 per cent. of the persons operated on recovered. Only 46 of the 236 recovered after the twelfth hour. The author calls attention to the occurrence of multiple perforation.

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[10] Cartwright Lectures.

It follows that skill in early recognition is essential to give the patient as well as the surgeon a chance. The medical attendant who recognizes the accident and calls the surgeon early is more deserving of honor than the operator, remarks a distinguished German surgeon.

It is important to note that the operation for perforation is followed by more substantial cures than any other procedure, that is, a permanent cure, hence fewer recurrences follow. French (St. Mary's Hospital) reports 30 cases operated on for perforation, 18 of which were followed many years, and 15 remained perfectly well. Gosh (St. Bartholomew's) notes 69 perforations—34 died—mortality, 49.2 per cent.; 11 operated on and 9 perfectly cured. Patterson's statistics do not fully uphold this view and show the frequency of recurrence after operative procedures in general. 35 patients were operated on for perforation. After fifteen months to two years 18 were perfectly well (16 ulcer closed and 2 closed and gastro-enterostomy). Recurrence took place as follows: 1 died of second perforation twenty-one months later; 2 required gastro-enterostomy; in 9 symptoms of gastric ulcer arose, and 5 had dyspepsia.

If our statistics are upheld it would seem that an operation which destroys the ulcer, as excision, would be ideal. This is the contention of Rydygier, who advocates resection. He lost 2 cases after gastro-enterostomy from perforation and two from hemorrhage and suggests that these complications are not infrequent. Professor Dock, in his Yale address, advocates a similar method of operation, and Bell of Minneapolis is also insistent upon this method, while Rodman earnestly urged excision of the ulcer-bearing area.

The Sequels. In the treatment of the sequels or complications of gastric ulcer the surgeon has equal domain with the physician. The following statistics will show how prominent a surgical affection it becomes, and how necessary it is to employ operative procedures. They show that it is not for gastric ulcer most operations are performed, but for the results of ulceration.

That the sequels of gastric ulcer which cause retention demand surgical treatment our statistics uphold, notwithstanding the opposition of weighty authority. This, however, must be said, the presence, and operation for the relief, of such sequels do not remove the patient from the category of medical supervision. It is as important to continue dietetic, hygienic, and medical means as formerly. Indeed, we must be willing to admit that all surgical procedures must be looked upon as those of necessity.

Cases from the Literature. Number of sequels, 766 (40.0 per cent.). Retention from stenosis, 504 (65.7 per cent.); adhesions, 156 (20.3 per cent.); hour-glass, 81 (10.5 per cent.); dilatation, 13 (1.7 per cent.); pylorospasm, 17 (0.91 per cent.); cause not stated, 5 (0.65 per cent.).

Duration of Symptoms of Sequels:

	Adhesion.	Stenosis.	Dilatation.	Pylorospasm.	Hour-glass.
1–6 months	4	24	0	2	1
6–12 months	13	46	0	0	8
1–5 years	44	92	4	2	19
5–10 years	46	127	5	1	22
10–15 years	20	52	2	1	12
15–20 years	14	43	0	1	9
Over 20 years	9	21	1	0	5
Not stated	6	99	1	0	5

This table shows the duration of symptoms prior to operation. The records in which the maximum number of sequels occurred is given below: Adhesions: Greatest number (43) between ages of 30 to 40 years. More females than males. Stenosis: Greatest number (118) between 30 to 40; more males than females. Hour-glass: Greatest number (22) between 40 to 50; more females than males. Pylorospasm: Three between 40 to 50, all females, and two between 50 to 60, all males. Dilatation: Three between 20 to 30, were females; and three between 40 to 50 were males.

All admit that ulcer is a disease of early life. The above statistics show, as we would expect, that the sequels are of late life. The duration of the symptoms, 5 to 10 years, upholds this. If the ulcer was diagnosticated early and treated properly, the late sequels would be avoided.

THE RESULT OF THE TREATMENT OF SEQUELS (CASES FROM LITERATURE).

	Medical.				*Surgical.*					
	Cured	Improved	Unimproved	Died	Cured	Improved	Unimproved	Died	Recurrence	Result not given
Stenosis	0	2	0	4	340	13	8	42	9	18
Adhesions	0	1	0	0	127	4	2	16	5	1
Hour-glass	0	0	2	0	61	3	0	12	3	..
Dilatation	2	0	0	0	7	3	0	1	..	..
Pylorospasm	0	0	0	0	6	0	0	1	..	2

DILATATION—13. (1.7 per cent.)

			Per cent.
Surgical	Cured	7	53.0
	Improved	3	23.0
	Unimproved	0	
	Death	1	7.7
Medical	Cured	2	

PYLOROSPASM—7. (0.91 per cent.)

Cured	6	85.7
Death	1	14.3

STENOSIS—504. (65.7 per cent.)

Surgical	Cured	340	67.3
	Improved	13	2.5
	Unimproved	8	1.5
	Death	42	8.3
	Not given	86	17.0
Medical	Improved	2	
	Death	4	

ADHESIONS—156. (20.3 per cent.)

			Per cent.
Surgical	Cured	127	81.4
	Improved	4	2.5
	Unimproved	2	1.2
	Death	16	10.2
	Not given	1	
	Recurrent	5	3.2
Medical	Improved	1	

HOUR-GLASS—81. (10.5 per cent.)

Surgical	Cured	61	75.3
	Improved	3	3.7
	Unimproved	0	
	Death	12	14.8
	Recurrence	3	3.7
Medical	Unimproved	2	

PERIOD UNDER OBSERVATION AFTER CURE. PATIENTS REMAINING WELL.

	Stenosis.		Adhesions.		Hour-glass.		Dilatation.		Pylorospasm.	
Time.	Surg.	Med.	Surg.	Med.	Surg.	Med.	Surg.	Med.	Surg.	Med.
1–6 months	70	2	31	1	12	..	3	1	2	..
6–12 months	59	..	23	..	17	..	3	..	..	..
1–2 years	69	..	15	..	4	..	1	..	2	..
2–3 years	20	..	5	..	4	..	..	..	..	..
3–4 years	13	..	4	..	5	..	..	..	..	..
4–5 years	6	..	1	..	1	..	..	..	..	..
Over 5 years	9	..	1	..	2	..	..	..	..	..
Not stated	106	..	51	..	19	..	3	1	..	..

The sequels, it is well seen, when they produce symptoms that are intolerant or perilous, are surgical affections. The nature of the lesion admits of no other conclusion.

The sequels in unpublished cases number 353. The lesions were not clearly defined or were confused in 53 which are not available for analysis. The greatest number were of 1 to 5 years' duration. All occurred late in life. The summary shows the decade in which the lesions were most frequent.

Adhesions. Largest number (13) between 30 to 40 years. More females than males.

Stenosis. Largest number between 30 to 40 years. More males than females.

Hour-glass. Largest number (4) between 30 to 40 years. All females.

Pylorospasm. Largest number (5) between 20 to 30 years. Seven males; six females.

Dilatation. Largest number of cases, equally divided between 20 to 30 and 30 to 40 years, nine males.

DURATIONS OF SYMPTOMS OF SEQUELS (UNPUBLISHED CASES).

	Adhesions	Stenosis	Dilatation	Pylorospasm	Hour-glass
1–6 months	5	8	18	8	..
6–12 months	8	31	15	7	..
1–5 years	23	77	34	21	4
5–10 years	10	47	18	3	1
10–15 years	2	8	2	2	1
15–20 years	1	5	2	..	1
Over 20 years	..	4	..	..	..
Not stated	2	2	6	4	1

Result of Treatment of Sequels (Unpublished Cases).

	Medical				*Surgical*					Result
	C	I	U	D	C	I	U	D	Recur.	not given.
Stenosis . . .	2	4	2	0	131	5	2	18	7S* 7M*	1 Med.
Adhesions . .	0	1	1	0	40	2	2	6	4S*	1 Med.
Hour-glass . .	0	0	0	0	6	0	0	1	1	
Dilatation . .	49	25	0	0	20	2	4	4	1S*	3
Pylorospasm .	25	6	1	0	9	4	1	1	1S* 1M*	

* S, surgical treatment; M, medical treatment.

Period of Observation after Treatment of Cases that were Cured, during which Time no Recurrence.

	Stenosis.		Adhesions.		Hour-glass.		Dilatation.		Pylorospasm.	
	S*	M*	S*	M*	S*	M*	S*	M*	S*	M*
1–6 months . . .	17	2	7	..	1	..	1	16	2	6
6–12 months . . .	11	..	4	..	..	..	3	8	4	4
1–2 years	12	..	4	..	..	..	2	4	..	4
2–3 years	10	1	5	..	..	..	2	13	1	8
3–4 years	5	1	3	..	..	..	..	2	..	..
4–5 years	5	..	2	..	..	..	..	..	1	3
Over 5 years . . .	3	..	0	..	..	..	1	1	..	4
Time not stated . .	76	2	17	1	5	..	13	19	5	2

* S, surgical treatment; M, medical treatment.

The difference between the cases published and those unpublished is notable in the greater number due to dilatation and to adhesions in the latter. This is due to greater care in giving the cause of stenosis. The surgical treatment of stenosis is attended by a high mortality, but it is presumed the cases were of very grave type and death would have resulted had not interference been permitted. In dilatation the mortality was higher with surgical than with medical treatment. Both forms of treatment were followed by the same percentage of cures. As with the published cases the age and sex were not factors determining the result, apart from their influence in all surgical procedures. It is this class of cases, we must agree with Greenough and Joslin, "which indicates the need of surgical intervention in other than emergency cases of the disease."

Frequency and Mortality of Operations. The statistics relating to the various operations present interesting features. The first of the group of tables shows the frequency of gastro-enterostomy and the conclusion therefore, of surgeons, that it is the operation to meet most of the difficulties. The percentage of mortality in gastro-enterostomy is much higher than that of individual operators. Mayo Robson has had a mortality of less than 5 per cent., and later, in ulcer, of 1.7 per cent.; Moynihan (1906) of 3.5 per cent.; Mayo of less than 3 per cent. If a number of operators are grouped, as by Joslin, the mortality is 23.8 per cent., a not wide departure from our statistics.

The mortality in private or unpublished cases, 9.1 per cent., approaches that of single operators. The mortality of excision is high and yet compares favorably with that of gastro-enterostomy, for recurrence took place in 58 of 964 cases of gastro-enterostomy, or 6.23 per cent., while after excision recurrence took place in 2 of 35 cases, or 5.74 per cent. 2 of the 9 of Mayo Robson's cases relapsed or recurred and 1 was not traced.

The percentage of death, when all the cases of all operators are grouped together, is higher than that of individual operators of large experience. It is well that this is known, for the surgeon who by design operates once or twice only should know the greater mortality. If any blackness can be given to statistics to deter the surgeon who is rushing in to cure dyspepsia, etc., it is also well.

NATURE AND FREQUENCY OF OPERATIONS.

Cases collected from the literature. Total number of operations, 1193.

		Per cent.
Gastro-enterostomy	964	80.80
Pylorectomy	24	2.10
Excision	51	4.20
Pyloroplasty	69	5.20
Cauterization	5	0.41
Dilatation of stricture	4	0.33
Gastrolysis	37	2.20
Suture	38	2.30

RESULT OF VARIOUS OPERATIONS (CASES COLLECTED FROM THE LITERATURE).

	Gast'ent.	Exc.*	Pyl'rec.	Gas.	Dil.	Caut.	Pylor.	Sut.
C	690	35	21	22	4	3	48	25
I	47	1	1	4	..	..	4	1
U	25	1	..	6	..	..	2	..
D	144	10	2	2	..	2	6	6
R	58	2	..	3	..	..	9	6

* In two cases of excision final result not given.

PERCENTAGE OF CURE OF VARIOUS OPERATIONS.

		Cured or improved. Per cent.	Unimproved or death. Per cent.
Gastro-enterostomy	964	76.4	23.6
Excision	51	70.6	29.4
Pyloroplasty	69	75.3	24.7
Cauterization	55	60.0	40.0
Dilatation of stricture	4	100.0	....
Gastrolysis	37	70.2	29.8
Suture	38	68.4	31.6
Pylorectomy	24	91.6	8.4

LATE RESULTS OF VARIOUS OPERATIONS (LITERATURE).

Permanency of cure in various operations.

	Gastro-enterostomy.				Excision.				Gastrolysis				Pylorectomy.			
	C	I	U	D	C	I	U	D	C	I	U	D	C	I	U	D
6 months	126	9	2	144	10	1	..	10	7	3	6	2	5	1	..	2
6–12 months	103	8	2	..	3	..	..	..	5	..	..	..	3	..	..	..
1–2 years	123	7	4	..	6	..	..	..	3	1	..	..	3	..	..	..
2–3 years	36	3	1	..	2	..	..	..	..	..	..	..	2	..	..	..
3–4 years	26	3	..	..	..	..	..	..	1	..	..	..	..	..	..	..
4–5 years	12	1			1		1		1							..
Over 5 years	18	2			2			..	..	..	..	..	..	..	..	..
Time not given	246	14	16		11	..	..	2	5	..	..	..	8	..	..	..
Recurrence	58	..	..	..	2		..	..	3	..	..	..	..	..	..	..

LATE RESULTS OF VARIOUS OPERATIONS (LITERATURE).—(*Continued.*)

Permanency of cure in various operations.

	Suture.				Dilatation.				Cauterization.				Pyloroplasty.			
	C	I	U	D	C	I	U	D	C	I	U	D	C	I	U	D
1–6 months	13	..	..	6	1	..	..	..	..	..	..	2	17	2	2	6
6–12 months	4	..	..	..	..	..	..	..	..	..	..	..	9	..	..	..
1–2 years	2	..	..	..	..	..	..	..	..	..	..	..	8	..	..	..
2–3 years	..	..	..	..	1	..	..	..	..	..	..	..	2	1	..	..
3–4 years	1	..	..	..	1	..	..	..	..	..	..	..	8	..	..	..
1–5 years	..	1	..	..	..	..	..	..	..	..	..	..	1	1	..	..
Over 5 years	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Time not given	5	..	..	..	1	..	..	..	3	..	..	..	5	..	..	..
Recurrence	6	..	..	..	..	..	..	..	..	..	..	..	9	..	..	..

OPERATIONS (UNPUBLISHED CASES).

Operations for simple ulcer, sequels, and complications. Total number of operations, 292.

		Per cent.
Gastro-enterostomy	264	90.4
Excision	7	2.3
Pylorectomy	12	4.1
Cauterization	1	0.3
Dilatation of stricture	0	0.0
Gastrolysis	5	1.6
Suture	0	0.0
Pyloroplasty	3	0.1

RESULT OF VARIOUS OPERATIONS (UNPUBLISHED CASES).

	Gastro-enterostomy.*	Excision.	Pylorectomy.	Gastrolysis.	Dilated.	Cauterize.	Pyloroplasty.†	Suture.
Cured	208	4	8	3	..	1	2	..
Improved	8	..	..	1	..	..	..	..
Unimproved	6	..	..	1	..	..	..	..
Death	24	2	4	..	..	..	1	..
?	1	..	..	..	..	..	..	..
Recurrence	16	1	..	..	..	..	..	..

* In one case of gastro-enterostomy result not given. †Gastroplasty, 1 cured; 1 death

	Per cent.	Per cent.	Per cent.	Per cent.
Gastro-enterostomy	C. 75.5	I. 3.0	U. 2.2	D. 9.1

Recurrence, and result not given; in remaining cases 10.2 per cent.

	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Excision	C. 57.1	I.	U.	D. 28.5	R. 14.2
Pylorectomy	C. 65.0	I.	U.	D. 35.0	
Gastrolysis	C. 60.0	I. 20.	U. 20.	D.	
Cauterization	C. one case				
Pyloroplasty	C. 66.6	I.	U.	D. 33.3	

LATE RESULTS OF VARIOUS OPERATIONS (UNPUBLISHED).

Permanency of cure in various operations.

Gastroplasty, 1 cured, 1 death.

	Gastro-enterostomy.				Excision.				Gastrolysis.				Pyloroplasty.			
	C	I	U	D	C	I	U	D	C	I	U	D	C	I	U	D
1–6 months	25	1	6	24	1	..	..	2	..	1	1	..	1	..	..	1
6–12 months	20	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..
1–2 years	15	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
2–3 years	13	1	..	..	..	..	..	..	1	..	..	..	1	..	..	..
3–4 years	5	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
4–5 years	5	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..
Over 5 years	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Not given	108	5	..	..	2	..	..	..	1	..	..	..	..	..	..	..
Recovered	16	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..
Recurrence not given	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..

In the remaining operations the data were not sufficient to make any calculations.

CONCLUSIONS. Gastric ulcer is a medical disease.

Gastric ulcer with complications and sequels is sometimes a surgical disease; if perforation occurs it becomes a surgical affection at once; if hemorrhage occurs acutely, it is rarely a surgical affection; if repeated and chronic, it is a surgical affection.

If the ulcer is productive of perversion of secretory function alone, it remains a medical affection. Inasmuch as hyperchlorhydria is in part a neurosis, the scretory function can be balanced chiefly by medical, dietetic, and hygienic measures. Even if pyloric spasm attends the hypersecretion and hyperacidity it does not necessarily take the case beyond medical care. It is wrong to submit such patients to operation, unless motor disturbances become prominent.

If the symptoms and physical signs of retention from obstruction, dilatation, hour-glass contraction, or adhesions supervene and persist, the case is surgical.

If the symptoms of gastric ulcer become continuous in spite of medical treatment and incapacitate or threaten life, and if hemorrhage recurs and secondary anemia arises, it is a surgical disease. Such cases, however, are always attended by organic sequels.

The extraordinary frequency of chronic gastric ulcer with sequels requiring operation is due to neglect of the treatment of an ulcer in its incipiency. Statistics show that most patients are operated on between the thirtieth and fortieth year and have an ulcer history of five or ten years' duration.

What, as a medical attendant, should one do with a case of gastric ulcer? From personal experience and a study of recorded cases I would say, if it is simple uncomplicated ulcer, employ rest, at first absolute and later modified, a suitable diet, and the drugs indicated, for at least four months. If attended by an organic complication, as pyloric obstruction from thickening or from adhesions, or by dilatation, if extreme, or by hour-glass contraction, surgical measures are in order.

If perforation exists there should be no delay in operating.

If hemorrhage exists operation is rarely necessary, and if acute, not unless the peril of hemorrhage outweighs that of operation—a nice estimation of values. If hemorrhage is persistent and gives rise to anemia, operation is indicated. Under any circumstances and until cure is established keep the patient in touch with a surgeon. The physician should never assume the attitude of a distinguished physician who congratulated himself that he did not ask a surgeon to see a case because it had features like those of pancreatitis, a suspicion borne out by the autopsy which showed such lesion. It should be the duty of the physician to associate with himself a surgeon, to the end that accidents may be taken care of at once, and organic sequels relieved.

The final very serious duty is the selection of the surgeon. One who has good technical ability and has had considerable experience in gastric surgery should be selected. The operation even of gastroenterostomy is not trivial and requires the best service at command.

After the surgical procedures of necessity are carried out the patient must be treated medically. Medical treatment must be continued over a period of four months at least; hygienic and dietetic treatment over a period of years.

A patient who has had gastric ulcer should, for all time, observe the hygienic and dietetic rules which keep digestion to an approxmately normal state, which prevent anemia, and which, above all, so conserves the nervous system as to prevent neurosis.

THE INDICATIONS FOR, THE METHODS OF, AND THE RESULTS TO BE EXPECTED FROM, THE MEDICAL TREATMENT OF GASTRIC ULCER.[1]

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To assemble the evidence that recent experience makes possible in the medical treatment of peptic ulcer and the secondary results of this disease, and to point out what advance has been made, is the first task set us—a part of the subject that will be dealt with by my distinguished colleague, Professor Musser. To decide, in view of all the facts, the most judicious course to pursue, without attempting to press overmuch the importance of any particular method of treatment, is the second task, and to that I devote this paper.

Were we to permit ourselves to take a brief in championing some method with the zeal that success in a comparatively small group of cases sometimes suggests, we should undoubtedly fail in complying with the intention of the makers of this program. Above all things, it appears important at this juncture not to array against each other the comparative advantages of medical and surgical practices. Rather there should be made the deliberate effort so to view the matter that we may concur in deciding upon our place in the treatment of this disease.

We should exclude from consideration those ulcerative processes dependent upon special infections, like syphilis, tuberculosis, and pyemia, except when there can be shown to be a definite relationship between these and peptic ulcer.

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[1] An address delivered at the Seventh Triennial Congress of American Physicians and Surgeons, Washington, D. C., May 7, 8, and 9, 1907.

We should attempt to exclude, although perhaps unsuccessfully, certain cases that closely resemble in symptomatology peptic ulcer, but in which, despite the occurrence of pain, tenderness, spasm, vomiting, and hemorrhage, no ulcer actually exists. It is asserted in some quarters that these cases do, in fact, suffer from undiscovered ulcer or fissure through which the bleeding occurs. I deny that this is always true. First, because oozing of blood may be seen through the turgid, deeply congested mucosa that shows no lesion even under the magnifying glass. Second, because we know that mere superficial erosions or fissures in an otherwise healthy stomach are rapidly cured by nature, and, practically, are without symptoms.

Within these limitations there remain two more or less distinct divisions of peptic ulcer. First, the more frequent type, the acute, at least in the beginning, which occurs in younger patients and which appears somewhat more often in chlorotic young women; and, second, the type more commonly developing in older patients, especially men past middle life, often beginning as a chronic ulcer, and hence possessing a greater tendency to persistence. The difference in the history of these two types strongly suggests that their etiology is not identical, and yet, there is enough sameness in the processes to enforce the belief that in most respects they represent a common disease with the presence merely of a variation.

It should be strongly emphasized, if we are to understand the subject aright, that the stomach possesses an inherent and powerful resistance to accidental injury from ordinary causes. It does not readily succumb to infectious foods nor to wounds from foreign bodies.[2]

I have carefully studied, before and after gastrotomy, the stomach contents of a man who for years swallowed knife-blades screws, nails, and glass. There was evidently irritation, but, nevertheless, fair digestion of ordinary foods, and, on surgical exploration, while there were scars and areas denuded of mucosa there were no peptic ulcers, nor was there a history of ulcer.[3] Beaumont long since proved that the gastric mucosa was repaired with surprising rapidity of injuries deliberately inflicted upon it. There is other evidence that may be adduced to show that the stomach not only resists injury successfully, but that it heals spontaneously in excess of other structures of the body.

From this we must infer that a peptic ulcer includes in its course and its nature some other factor and quality than a local injury, infection, and an excessively acid gastric juice. We may not exclude these as elements in the cause; they are but steps in a series of which the chief is some influence that removes from the mucosa its inherent resistance—something that, when present, enables it to endure a hyperchlorhydria; when absent, leaves the mucous membrane and the other gastric layers an easy prey to the digesting, eroding, gastric juice.

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[2] It is interesting to note how the lower animals gorge themselves with putrefied animal tissue, fragments of bone, and irritating substances without apparent injury to the gastric mucosa, although, in the case of dogs, the gastric acidity is extremely high.

[3] This was a case of Dr. Gaylord, who made a gastrotomy, and removed 577 foreign bodies of various kinds, besides 144 grams of broken glass, many fragments of which were embedded in the gastric mucosa. Numerous pieces of mucous membrane were separated from the stomach in the efforts to remove the foreign bodies. The patient made a good recovery, and there was little difference in the gastric digestion before and after the operation.

That this must be true is evidenced in the instances of serious, even fatal, ulcer in patients having gastric juice of low acidity and feeble digestive power, and, conversely, by the numerous examples of hyperchlorhydria with exemption from ulcer.

The preventive treatment, the immediate treatment of necessity and the after-treatment in surgical cases should take cognizance of these important facts. It is because we have not solved this perplexing, but perfectly obvious, problem that we find the embarrassing recurrences both after medical and surgical treatment. For it must be admitted that the knife has not proved to be the panacea in gastric ulcer that it has in appendicitis; and physicians readily consent to the statement that the medical treatment of chronic cases in old men is often unsatisfactory, and in a certain percentage ends in failure.

At least our attitude toward treatment should embrace the acceptance of the reality of a definite lowering of internal cellular resistance in some focus or in foci of the gastric mucosa—points that are more often located near the pylorus, at the lesser curvature, posteriorly.

Weinland believes that in the cells of the gastric mucosa is developed a substance, an antibody, that antagonizes the digesting effect of the ferments. This substance may be absent or much decreased in the focus wherein peptic ulcer develops.[4] Why this local deficiency? It has been suggested that it follows a nerve lesion, and some have emphasized that it probably depends upon a trophoneurosis. As herpes facialis appears in certain individuals with a slight general infection, after mental or bodily fatigue (and herpes occasionally persists until the general health is restored), or as painful perforating ulcer of the arm or leg, or as a hematoma auris of the insane, may, and apparently do, depend upon a neurotrophic disturbance, so may not the weakness in tissue resistance of the stomach, including the disappearance of antibodies, be likewise accounted for? Granted the actual presence of this local tissue depression, no matter what its nature, we can understand how increased activity, spasm, coarse and irritating foods, external pressure, etc., come to assist in the evolution of gastric ulcer. Eiselsberg, of

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[4] C. Bolton. Trans. Royal Soc., September 28, 1904, describes a specific gastrotoxic serum containing an agent that produces necrosis, and another that produces hemorrhage in the gastric mucosa.

Vienna, advocates and practises the making of a jejunal fistula through which the patient may be abundantly fed. Of course, this establishes gastric rest, but Eiselsberg[5] believes that the improvement in the general nutrition of the patient also contributes considerably to the cure as well as to the prevention of recurrence.

Our preventive treatment, then, should be so to guard the patient that the nervous system suffers least strain, that the gastric juice may be moderated in its activity, and that the gastric motility (through improper food, stimulants, etc.) be not exaggerated.

The prevention of gastric ulcer might be summarized thus: maintain a calm nervous system in a well-conditioned body. It has been shown that chlorosis is accompanied by gastric hyperacidity; hence anemia must be overcome. The sharp seeds of fruits, the resisting portions of vegetables rich in cellulose, nuts, salads, condiments, etc., call for unusual, often excessive motor activity of the stomach, and hence should be excluded from the diet.

In acute gastric ulcer the great need is for early dagnosis, for positive conviction, for strict discipline in managing the hygiene, the diet, for relieving the hyperchlorhydria when present, as it usually is, and for prolonged treatment. When this control is righteously observed, there is, as a rule, little trouble in bringing about immediate relief of symptoms and eventually inducing cure of the disease. The unfortunate cases are those wherein diagnosis is uncertain, or the treatment careless, halting, and incomplete. Under such circumstances it is to be expected that a certain percentage will become chronic cases, and a small percentage perforative cases. The fault is not in the possibilities of medical treatment but in its indifferent application. Even in cases in which hematemesis is early and severe this statement holds true.

If in acute hemorrhage the surgeon intervenes and performs a gastro-enterostomy, he is not sure of saving the patient. Indeed, experienced surgeons have informed me that they consider this operation unsuited in acute cases with hemorrhage. It would seem to me an unsafe reliance unless the ulcer area be at the same time attacked. Such an operation would seem to involve a grave risk; far greater than proper medical treatment for stopping bleeding. Nevertheless, in very rare cases in which medical means prove unsuccessful and death is imminent, we should consent to give surgery a trial; but it is somewhat unfair to surgery to expect sure relief in this serious state of affairs.

What medical treatment should be adopted in acute cases with bleeding? Procure the most complete rest obtainable of body and mind and keep the stomach out of function. Sometimes most satisfactory results are obtained by carefully, and very rapidly, washing out the stomach with ice-water,[6] using a large, soft, blunt-pointed stomach tube, or by introducing a weak solution of adrenalin. When the bleeding has ceased, wash out the adrenalin solution and pour in a small amount of gelatin solution, quiet the patient with morphine[7] and atropine or hyoscine; allow him frequently to take bismuth and light magnesium carbonate, suspended in gelatin water, provided there is over-acidity; later introduce (per rectum) frequently small quantities (30 to 60 c.c.) of warm, normal saline solution. Such measures usually control even profuse bleeding.

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[5] Wiener klin. Woch., 1906, Nr. 43, p. 1298.

[6] Ewald, Wien. klin. Woch., 1906, No. 12.

Thereafter there should be rest without food, except the saline enemas, for several days—a week, perhaps. Should we nourish per rectum? Not at first; for the reason that nutrient enemas excite gastric secretion. After two or three days, or immediately, if needed to prevent collapse, small nutrient injections of milk and glucose should be used. As a rule, the intelligent use of normal saline solution per rectum, small frequent injections, will accomplish more than nutrient enemas, especially early in the case. When hemorrhage persists or threatens, chloride of calcium in doses of 1 to 2 grams, diluted as necesssary, may be alternated with the sodium chloride solution.

When should we feed the patient? It is better to enforce absolute rest of the stomach for a period of from three to six days. Should alarming weakness appear, aliment should be given. This should be in the form of a thin gruel of arrowroot, farina, etc., perhaps blended with a little lime water and milk, or, if it appears to agree better, egg albumin in water or expressed beef juice, at first in small quantities (liquid,) unstimulating, and always with close watching of results. If the patient endures the starvation well, let him wait five or six days and then begin feeding small portions of milk or gruel, gradually increased. After a few tentative days, if all goes well, larger amounts should be allowed.

Here arises the question as to which is best,—a scant fare, full fare, nitrogenous or non-nitrogenous foods. A study of the effect of quantity and nature of the food allowable in improving gastric ulcer leads to the conviction that each case must be a law unto itself. One is unwise to insist upon a rule. He should be modest and study his cases carefully. One patient will do best on rather early and rather full feeding, as recommended by Lenhartz; another becomes distinctly worse until complete rest of gastric activity has been carried out for several days, as practised by von Leube. It seems to me that these contradictions are to be explained by the element of gastric and pyloric spasm that plays such an important role in this disease. Hence the question of excitability, of central and local irritability, needs most thoughtful study. Spasm must be prevented, and thereby motor insufficiency relieved, so that free drainage may be effected. If the results are compatible with feeding, the patient should be fed; if made worse by feeding, he should be starved. I am convinced that in suitable cases one need not fear allowing a rather full amount of carefully divided and broken-up animal food (such as meat juice, ground meat, raw eggs, milk preparations, etc.), beginning with small amounts and gradually increasing the quantity, avoiding the factor of stimulation and overcoming any symptoms of hyperacidity by administering, as required, a mixture of cerium oxalate (1 part), bismuth subcarbonate (2 parts), and light magnesium carbonate (4 parts), or lime-water, milk of magnesia, and permitting the drinking of warm alkaline water. On the other hand, when there is marked motor excitability, with a tendency to pyloric spasm that seems to be increased even by bland liquid foods, complete abstinence must be observed for several days. Meantime the external application of poultices, or of cold fomentations, as advised by von Leube, are distinctly useful. The good effect of these external applications may be explained in the soothing action which they exercise upon the motor excitability of the stomach. Provided the organ can be kept in a state of relatively passive relaxation, the stomach will empty itself, and drainage will be secured.

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[7] Morphine has the effect of increasing gastric secretion, and hence should be combined with atropine.

This state of gastric calm is occasionally assisted by the giving of fresh olive oil or unsalted butter. Orthoform or anesthesine are not only useful in diagnosis, as pointed out by Murdock, but also in the control of pain and indirectly of spasm.

Let us insist and teach that patients with peptic ulcer should submit to early and long-continued medical treatment, that they are not cured merely because the symptoms have disappeared, and that they be made to return from month to month, or from week to week, for observation, and at times for thorough examination, including the search for occult blood. In this way only, as indicated by Billings, can we be reasonably sure of good results.

From the medical point of view, surgery is called for in ulcer cases to relieve secondary results; that is, to save life when perforation occurs; to secure drainage when stasis of the second degree exists, and when it is not relieved by medical treatment; to stop otherwise uncontrollable bleeding; to overcome stenosis or the results of perigastritis, such as disturbances of the motor function from adhesions; and to relieve continued severe pain.

To this extent, the indications for surgery are for relief of results of gastric ulcer rather than for the cure of the disease itself. For the cure of the disease in obstinate cases the plan followed by Eiselsberg, which has already been referred to, seems reasonable and may yet win for itself general approval. Except when stenosis exists, gastro-enterostomy alone appears not to be positively curative of peptic ulcer, notwithstanding its usefulness in relieving symptoms. Physicians oppose the operation because it is too unphysiological, because it does not spare the important functions of the pylorus and duodenum, and because patients subsequently suffer from digestive symptoms, the recurrence of the ulcer, and even the starting of new peptic ulcers in the intestine.

To summarize the medical treatment:

Attempt to secure a calm mind, a quiet nervous system, and improvement of the general health.

Make a positive diagnosis, begin treatment early, and carry it out with painstaking attention to details for a long time.

Obtain general rest. In some cases feed the patient sufficiently, but discretely; in others, starve the patient for a period, depending for support upon frequent, small enemas of normal salt solution.

For the control of hemorrhage, in addition to rest, one may succeed by local treatment through the stomach tube, using ice-water, or adrenalin solution, followed by gelatin water.

In irritating hyperacidity, one should use local general sedatives and antacids.

To relieve hypertension and spasm of the stomach, in addition to suitable drugs, use external applications according to von Leube, or the equivalent of these.

Finally, continue treatment long after apparent cure, and study the stools for occult blood; but we should also remember the dictum of Bettmann[8] that, "it is not alone the question of how long treated, but how well treated." Statistics mean little, because of the uncertainty of method and the faulty detail so often observed in the treatment of this disease.

STUDIES ON ARTERIOSCLEROSIS, WITH SPECIAL REFERENCE TO THE RADIAL ARTERY.

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Among the first things which we record in the every-day examinaton of a patient is the palpability and consistency of the radial artery. While there has been much discussion as to what inferences one is justified in drawing with regard to the condition of the general arterial tree or of special central vessels, from the condition of the radial, we have, as a rule, little conception as to just what we are feeling in the radial artery itself.

In order to gain information upon this point one of us (Fabyan)

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[8] New York Med. Jour., March 9, 1907.

He is to be credited justly with the development of a splendid technique and with the reduction of operative mortality to a very small figure. But as to the ultimate result there remains some doubt. When we hear of a surgeon operating on a thousand or more cases and a score or more surgeons reporting their hundreds, the thoughtful observer, and especially the internist of experience, pauses to inquire: Is surgery successful because the internist has been lacking in his efforts as to general treatment? An answer to this question is difficult. One should know the course and death rate of exophthalmic goitre and of a variety of complicating conditions. These are lacking. The fact, however, remains that the problem is not the reduction of the death rate, but the prevention of invalidism, for such statistics as are available give the impression that death from exophthalmic goitre is not a frequent occurrence. The rate in medical practice is not much, if any, higher than the surgical death rate of three per cent. (this of course excludes death from mechanical causes which should never be allowed to occur). That the important problem is the avoidance of invalidism cannot be denied. In this connection, the details of the clinical histories of a few individuals, may be illustrative.

Case I.—A woman, now aged forty-five years, was at the age of twenty desperately ill with symptoms of extreme hyperthyroidism, and for months severe nervous phenomena compelled confinement in an insane asylum; at times death seemed imminent. After a year of general medical treatment complete recovery took place. Ten years later she was still in normal condition. Four months ago hyperthyroidism in pronounced form returned, but with treatment by rest and care all symptoms disappeared. She is now suffering from pronounced myxedema. This is the third case of such swing of secretory activity from plus to minus that I have seen. Would the myxedema have developed earlier had a portion of the thyroid gland been removed early in the disease? Do we know enough of the results of present day surgical treatment of exophthalmic goitre to know that this change is not a remote possibility of operative interference?

Case II.—When aged twenty years, Miss S. had pronounced hypertrophy of the thyroid gland with excessive secretion, characterized by such symptoms as tremor, tachycardia, nervousness, diarrhea, emaciation, and exophthalmos. After three years of continued treatment all symptoms of hypersecretion disappeared; the enlargement of the gland and exophthalmos remained. The patient has since married and has four children.

Case III.—A business man, aged fifty years, presented, in 1897, enlargement of the gland with extraordinary symptoms of hypersecretion. A vicious life and an unusually harrassing experience in the stock exchange preceded the symptoms and the history showed a possibility of the disease having first developed at the time of long residence in a goitre district of Michigan. The heart was very dilatable, the orifices enlarging and contracting from week to week; an aortic regurgitation appeared and disappeared. Many months of treatment were followed by a cure without return of symptoms until the present time.

Other histories might be quoted to show that it is wise to delay, or at least not to decide hastily, on the question of operation for endemic exophthalmic goitre. Many of my cases are too recent to report in final form, but my records show that with purely medical treatment a number have been cured and have remained well for six years or more.

Many minor points, it seems to me, are not infrequently ignored, or too lightly valued by advocates of surgical treatment. The latter is too frequently adopted without testing first the value of general treament. Also, it is sometimes of value to try the effect of drinking water different from that of the locality in which the patient has lived during the development of the disease, and again it is well to remember the relations, as emphasized in the earlier papers of McGraw of Detroit, which are now well nigh forgotten, of uterine and ovarian functions and of pregnancy to the secretion of the thyroid gland. My cases falling in this group are not many, but I have occasionally been impressed by the improvement of the thyroid condition after organic or functional disorders of the reproductive organs have been corrected. As an example of such improvement the following case is illustrative:

Mrs. L., five years ago, had a large fibrocystic goitre with hypersecretion and operation was necessary to avoid strangulation by mechanical pressure. Two years later the secretory symptoms returned associated with menorrhagia, and symptoms of prolapsus uteri. It was the general opinion of those who had seen the patient that it was wise to operate on the goitre and the pelvic disease at the same time. The writer believed that the latter operation would be sufficient and this alone was done; as a result all symptoms of goitre disappeared. Recently, however, after a period of a severe mental strain and anxiety the secretory symptoms recurred, but not with the same severity as before.

In a second case, Mrs. S., symptoms of marked hypersecretion of the thyroid were accompanied by a uterine disturbance, inflammatory in nature; treatment of the latter by a gynecologist led to disappearance of the pelvic trouble as well as the symptoms due to the thyroid.

After having observed many cases of exophthalmic goitre under various circumstances, the question to my mind is whether or not *any* violence to the system may not bring about temporary or even permanent relief of the symptoms. Thus, in one instance, the poor sufferer who had twice been operated on by Kocher, lost his eye by an explosion of fireworks. The eye had to be enucleated.

and roughened in outline and of a deep black hue. Gradually, the irregular, rough, deeply staining inner layer is at points lifted from the surface of the interna, forming a separate, more or less parallel

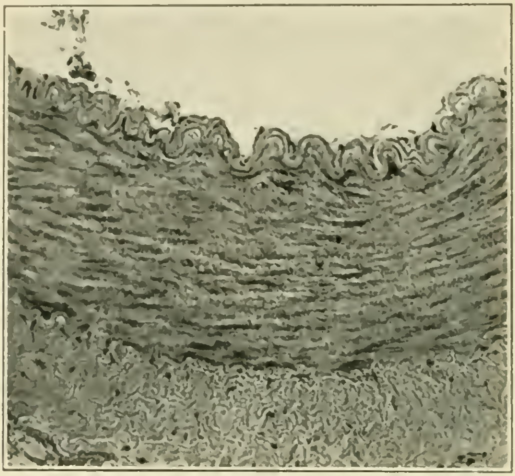

FIG. 4.—Radial artery of a child five years old (Case 61) who died of bronchopneumonia following measles (hematoxylin and eosin). This specimen shows a beginning elastic muscular thickening of the intima with the separation of a second layer of elastica from the surface of the intima. This artery was not palpable clinically.

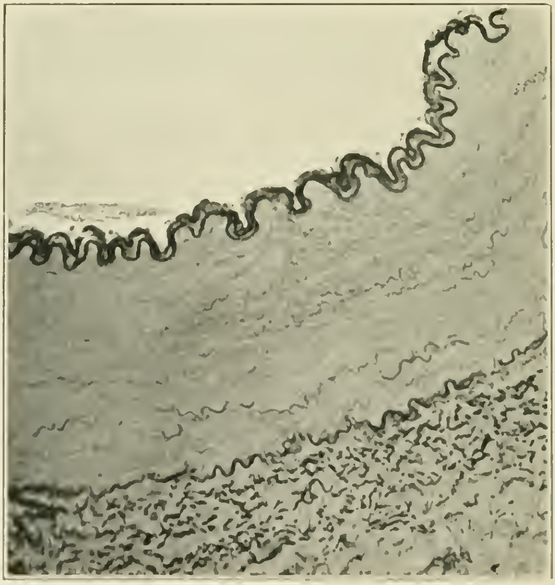

FIG. 5.—Radial artery of a child five years old (Case 61) who died of bronchopneumonia following measles (Weigert's elastic tissue stain). The specimen shows the early separation of an inner layer of elastica from the surfaces of the interna. This inner layer has at points a somewhat beaded appearance.

strand (Fig. 6). This strand on cross section appears beaded, and in longitudinal sections much smoother, justifying, in our opinion, the conclusions of Hallenberger,[1] that it represents a collection of longitudinal fibres. In some specimens, connective-tissue cells may be seen apparently making their way through the interstices of the fenestrated membrane, giving rise to the picture on which Hallenberger bases his hypothesis that ingrowth of connective tissue causes the separation of these inner layers of longitudinal elastic fibres. At the same time between these two layers, and sometimes between the inner layer and the endothelium there appear a few connective-tissue cells and smooth muscle fibres which have in the main a longitudinal course.

The *media* also increases in thickness with the growth of the individual. The elastica in this coat is very scanty in amount, merely an occasional thin wavy fibre. Just external to the elastica interna there may be a delicate layer of connective-tissue fibres.

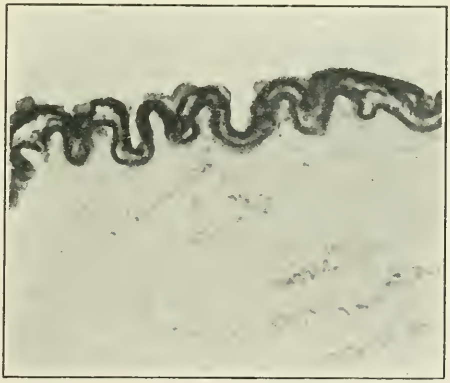

Fig. 6—Radial artery of a child five years old (Case 61) who died of bronchopneumonia following measles (Weigert's elastic tissue stain). The specimen shows the same changes noted in Fig. 5 under a higher power.

In the *second decade* the artery gradually assumes the character of the adult vessel; the walls become thicker but the vessel still lies collapsed like a ribbon on its muscular bed. The surface shows on section a number of delicate, transverse striations, as well as two longitudinal lines which correspond to the points at which the artery folds when in a state of collapse. The cause of the transverse striations is uncertain. Their appearance at the same time with the development of longitudinal elastic and muscular fibres in the intima, and the fact that they disappear when the artery is stretched, have suggested to us that they may be due to the contraction of these fibres.

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[1] Ueber die Sklerose der Arteria radialis, Marburg, 1906, 8. This valuable communication did not appear until some time after the beginning of our studies, and was unknown to us until our work was nearly done. It is satisfactory to find that our observations are in most particulars confirmatory of Hallenberger's results.

The *intima* has increased materially in thickness. A second strip of elastica is usually present. More, longitudinally arranged, smooth muscle cells have appeared as well as a small number of connective-tissue cells.

The *media* is also thicker (Fig. 7), the elastic tissue being relatively less marked and not as uniformly dispersed as in the first decade. Delicate fibrils running in various planes seem to arise from the interna and externa. No connective tissue is demonstrable excepting a fine layer just external to the interna.

In one case in this decade, a boy with chronic nephritis, there were local areas in which the intima was slightly more thickened (observable only by the microscope). In these areas, beside the second strand of elastica, there was a meshwork of finer elastic fibrils with an appreciable increase in connective tissue. Outside these points the elastica interna seemed less wavy and appeared rather stretched.

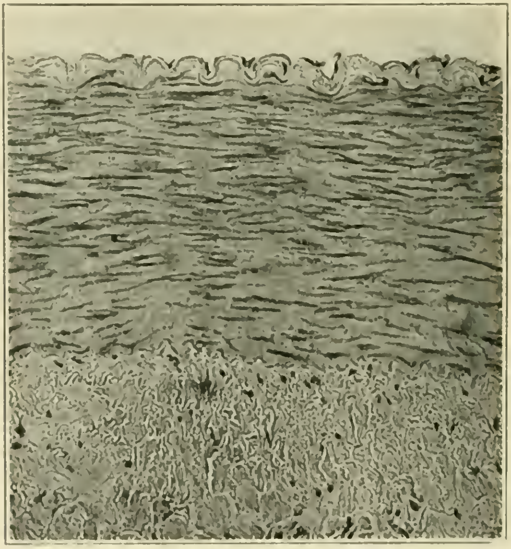

FIG. 7 Radial artery of a girl, aged fourteen years (Case 35), who died of pneumonia and pericarditis (hematoxylin and eosin). The specimen shows early elastic muscular thickening of the intima. This artery was just palpable clinically, but was not regarded as thickened.

In the *third and fourth decades* there is no great change in the constitution of the vessel. The *gross appearance* of the artery when laid open is the same excepting that in a few cases an occasional longitudinal line of irregular length is to be noted. The significance of these is not entirely clear, but their association, as a rule, with the presence of microscopic areas of connective-tissue thickening of the intima which contain numerous fine elastic fibres has suggested to us that they may be due to irregularities in the contraction of the vessel associated with these changes.

The *intima* and *media* tend to become somewhat thicker. The intima always shows a second strand of elastica and in several instances as many as two or even three additional strands. In addition to these, sometimes in irregular patches, sometimes more or less uniformly, the larger strands are replaced by a meshwork of fine fibrils with a marked increase in connective tissue (Fig. 8). These areas of connective-tissue thickening are especially noticeable in the vessels of a laborer, aged twenty-two years (No. 37), who had been a very heavy worker; of a laborer, aged twenty-five years (No. 60), with chronic nephritis; of a man, aged thirty-one years (No. 48), with aortic insufficiency; of a sailor, aged thirty-five years (No. 33), with mitral and aortic insufficiency and dilated heart. In one of these cases, that of the man, aged twenty-two years, there was a spot of calcification in the deeper part of a marked connective-tissue thickening of the intima (Fig. 9).

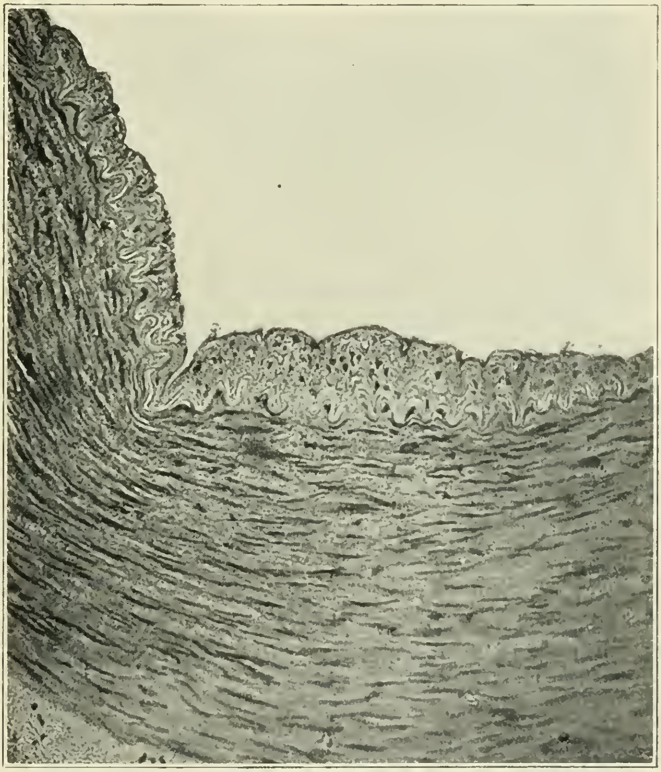

FIG. 8.—Radial artery of a healthy man, aged thirty-two years (Case 38), who died of a traumatic cerebral hemorrhage (hematoxylin and eosin). Elastic muscular thickening of the intima with a local sclerotic patch showing a fine elastic network. This artery was palpable clinically, but was not regarded as a thickened vessel.

The *media* becomes gradually somewhat thicker, and connective pregnancy, in all of which pregnancy produced a great increase in the severity of the disease. In 2 cases pregnancy was prolonged to its natural termination; in 2 cases labor was induced; and 1 case terminated by the spontaneous expulsion of a small and macerated fetus.

In the light of our present knowledge, Charcot's opinion that pregnancy sometimes improves these patients must be considered as erroneous. In Resinelli's experience a careful regulation of hygiene, the use of electricity, and especially the Röntgen rays, and the administration of antithyreodin are indicated; if evidence of kidney failure supervenes and the patient has reached the seventh month with a living fetus, labor should be promptly induced.

Skutsch[12] reports the case of a woman, aged thirty-nine years, in her fourth pregnancy, married five years. During her first labor the thyroid gland enlarged, but afterward diminished in size, and in the second and third labors was not perceptibly enlarged. In her fourth pregnancy exophthalmic goitre developed rapidly; the symptoms were relieved by large doses of bromide of sodium. Nutrition failed, and during the last weeks of pregnancy the patient lost strength and became melancholic, fearing that she would die as a sister had previously died from exophthalmic goitre. Labor developed spontaneously with the expulsion of dead twins, at about six months' development. There was little hemorrhage, but on the following day, after great restlessness and coma, the patient died.

Skutsch cannot believe that pregnancy in itself causes exophthalmic goitre. He believes that individual cases vary greatly as to the development of dangerous symptoms. There can be no question, however, but that the disturbance in metabolism caused by pregnancy is very dangerous to a patient having exophthalmic goitre, or having a predisposition to it. He considers the great disturbance in the action of the heart as especially dangerous. He positively denies the opinions previously advanced by Charcot, Carlein, Trousseau, and Sonza-Leibe, that pregnancy ever influences exophthalmic goitre favorably.

Massini,[13] of Buenos Ayres, has studied the influence of thyroid conditions upon eclamptic convulsions. His experiments show that while three-fourths of the gland may be safely extirpated in non-pregnant bitches, in pregnant bitches the same operation is followed by severe convulsions.

Ward[14] reviews extensively the literature of thyroid disease and reports fully the interesting case of a primipara at the end of the third month, married six years; two miscarriages, spontaneous at the third and fourth weeks. The family history showed exophthalmic goitre. The patient, when aged twelve years, had nervous symptoms of the disease manifested later on by tachycardia. This was improved by the use of cytotoxic serum and thyreoglobulin. Curetting was performed. This was followed by a marked return of the symptoms, which subsided under the serum treatment. Pregnancy ensued. The patient was decidedly nervous and restless, with moderate hypertrophy of the thyroid, constipation, and morning sickness. Injections of thyreoglobulin appeared to be beneficial until the end of the fifth month, when the urine greatly diminished, headache and vomiting becoming severe. In the sixth month a nitrogenous partition showed ammonia, creatinin, and rest nitrogen, greatly increased, with diminished urea. During the first two weeks of the sixth month the dose of thyreoglobulin was increased to 10 minims, twice daily; then three times daily, acting almost as a specific. The urine increased following the injections. During the seventh month the patient was very ill, with exhaustion and greatly diminished nitrogenous excretion. In the eighth month it was thought impossible for the pregnancy to proceed, and labor was induced by manual dilatation and bags. Dilatation being difficult, vaginal hysterotomy was performed, and a feeble child, weighing 6 pounds, was readily delivered by forceps. The placenta was adherent and was extracted. The patient rapidly recovered. The child lived thirty-six hours, dying from intracranial hemorrhage.

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[12] Zentralbl. f. Gynak., 1908, Band lxviii, p. 917.

[13] Paper published in Spanish and reprinted in L'Obstétrique, 1908, No. 5.

[14] Surg., Gyn., and Obst., December, 1909

Ward calls attention to the use of a saline extract of thyroid proteids made from fresh normal human glands as being more efficient than sheep thyroids, as ordinarily prepared. The hypodermic method he considers much superior to administration by the mouth.

Seitz[15] reviews previous experiments upon the relation between disease of the parathyroid bodies and eclampsia. His studies led him to conclude that during pregnancy the parathyroids become softened, more vascular, with an increase in the chromophile cells. In eclampsia these cells are lessened or disappear. Connective tissue increases, fatty colloid material and cysts develop. In some cases tuberculous infection has also been present. He believes that one must sharply distinguish between tetany in pregnant animals produced by partial or total removal of the parathyroids, and eclampsia. The so-called eclampsia of animals as seen in the cow he considers tetany and not true eclampsia. Nor does he believe that extract of the parathyroids can be successfully used in eclampsia.

Stowe[16] brings an interesting point into the study of this subject in reporting the case of a young woman in her second pregnancy, who a year previously had an abdominal section for acute appendibut the increase from the last decade is, relatively, considerably less than that of the intima. Opposite the thicker plaques in the intima the media is often considerably thinned, the nuclei elongated

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[15] Arch. f. Gynäk., 1909, Band lxxxix, Heft 1.

[16] Amer. Jour. Obst., May, 1909.

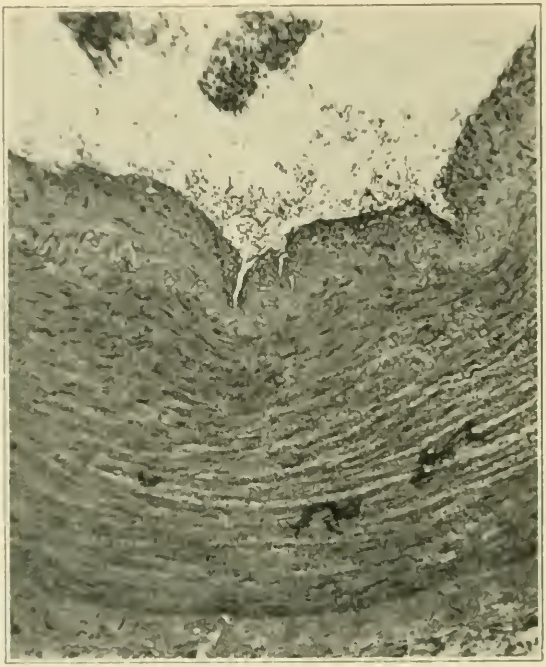

FIG. 11.—Radial artery of a day laborer, aged forty-one years (Case 54), who died of aortic insufficiency and pericarditis (hematoxylin and eosin). Diffuse sclerosis of the intima. This artery was regarded clinically as a thickened vessel.

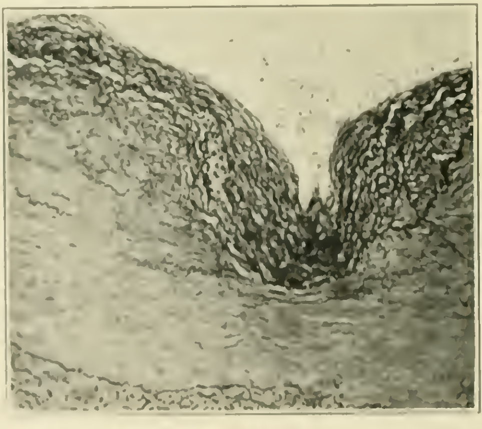

FIG. 12.—Radial artery of a day laborer, aged forty-one years (Case 54), who died of aortic insufficiency and acute pericarditis (Weigert's elastic tissue stain). Diffuse sclerosis of the intima. The elastica interna has disappeared. Meshwork of fine elastic fibres throughout the intima

narrowed, and diminished in number; there is a good deal of connective tissue. In 3 of those cases in which calcification was noted in the intima there was also a medial calcification, most marked in the inner part of the coat, close to the elastica interna, opposite the points of intimal calcification. In 1 instance it was very extensive, involving nearly the whole width of the coat in places, producing a picture similar to that described by Mönckeberg.[2] One can sometimes trace the deposition of lime salts directly into muscle cells from which the nuclei have disappeared, while in the periphery of these areas there may be evident hypertrophy of the individual muscle fibers. The tissue about the calci-

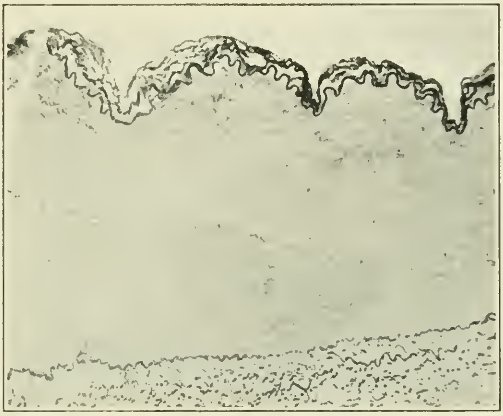

FIG. 13.—Radial artery of a day laborer, aged forty-two years (Case 44), who died of chronic nephritis (Weigert's elastic tissue stain). At points there are multiple strands of elastica. At other points there is a fine elastic network. In places the interna seems thinned and stretched. This artery was regarded clinically as a thickened vessel.

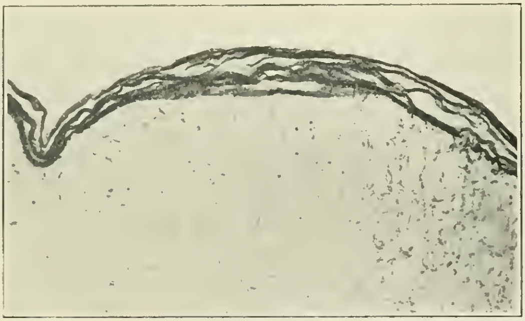

FIG. 14.—Radial artery of a day laborer, aged forty-two years (Case 44), who died of chronic nephritis (Weigert's elastic tissue stain.) Longitudinal section shows multiple strands of elastica in the intima.

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[2] Ueber die reine Mediaverkalkung der Extremitätenarterien und ihr Verhalten zur Arteriosklerose; Arch. f. pathol. Anat. u. Physiol. u. f. klin. Med., 1903, Band clxxi, 141.

fied areas has sometimes a homogeneous appearance staining with eosin, while the nuclei have disappeared.

The elastic tissue in the media is scanty—merely a few thin, straight fibres parallel, in cross sections, to the lumen. In this

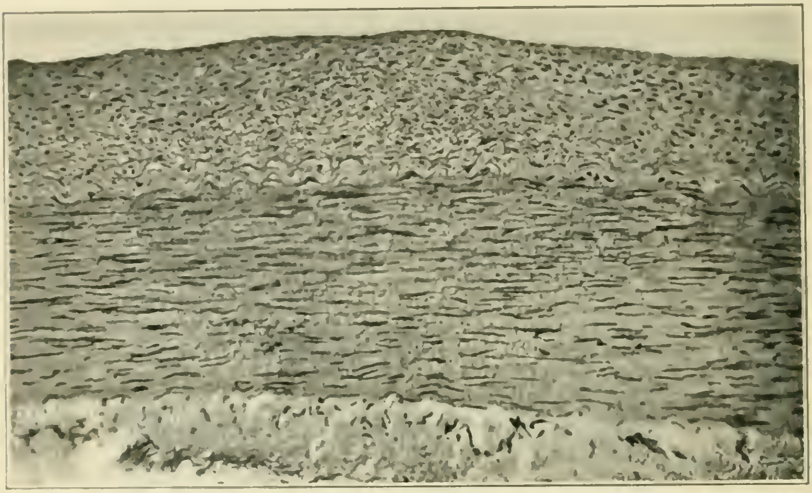

FIG. 15.—Radial artery of a tailor, aged sixty-four years (Case 49), with chronic nephritis and hypertrophied heart, who died of an œsophageal carcinoma (hematoxylin and eosin). Diffuse sclerosis of the intima. Clinically this artery was palpable and regarded as a thickened vessel.

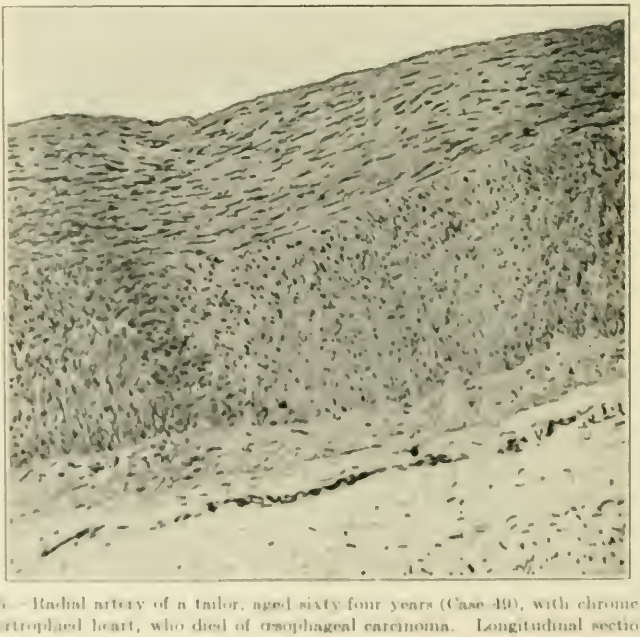

FIG. 16.—Radial artery of a tailor, aged sixty-four years (Case 49), with chronic nephritis and hypertrophied heart, who died of œsophageal carcinoma. Longitudinal section (hematoxylin and eosin). Diffuse sclerosis of the intima.

decade there were 6 cases in which the media was especially thickened. Of these, 3 were instances of chronic nephritis, 1 of aortic insufficiency, and 2 occurred in individuals who had been extremely heavy workers and alcoholics.

After the fifth decade there is a progressive increase in the thickness of the intima. From one to three, more or less regular elastic strips may be split off from the interna, but with advancing years these are less marked and a diffuse connective-tissue thickening of the intima with fine, irregular, elastic fibres becomes the common type (Figs. 15 and 16). Calcification in the deep layers of the intima (Fig. 17) becomes commoner with age, 4 out of 5 cases in the eighth and ninth decade showing this change.

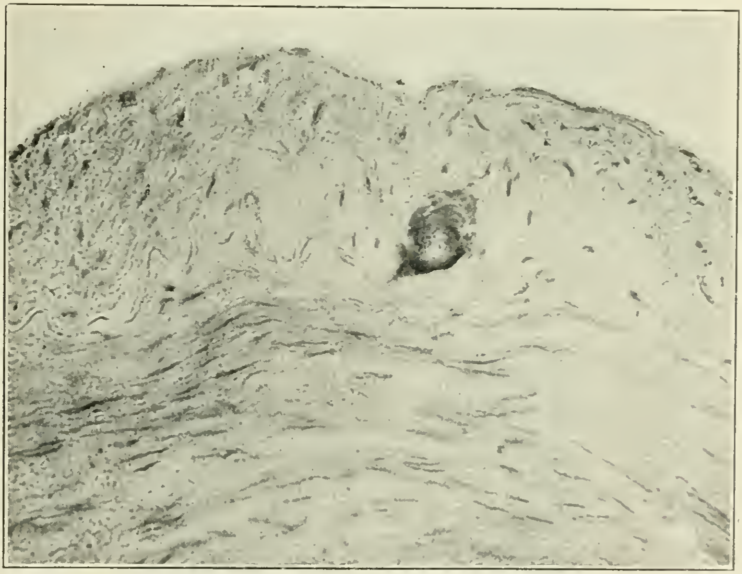

FIG. 17.—Radial artery of a carpenter aged sixty-six years (Case 50), with carcinoma of the stomach, who died of acute peritonitis; high power (hematoxylin and eosin). Calcified area in a sclerotic intima. This artery was clinically a thickened, beaded vessel.

The *media* after the fifth decade becomes, on the whole, rather thinner; there is a marked increase in the connective tissue and the tendency to calcification is greater. Opposite points of especial intimal thickening the media may be much thinned and evidently stretched, while the muscle fibres have in great part disappeared, having given way to connective tissue. The elastica interna in such cases is either much stretched or absent (Fig. 18). In some instances, as shown in the photograph of the artery of a man, aged eighty-three years, the whole vessel may be changed into an irregular, nodular tube (Fig. 19).

It thus becomes clear as pointed out by Jores[3] from a general standpoint, and by Aschoff,[4] and Hallenberger,[5] with regard to the radial especially, that an elastic muscular thickening of the intima appears in the radial artery at a relatively early age, that is, within

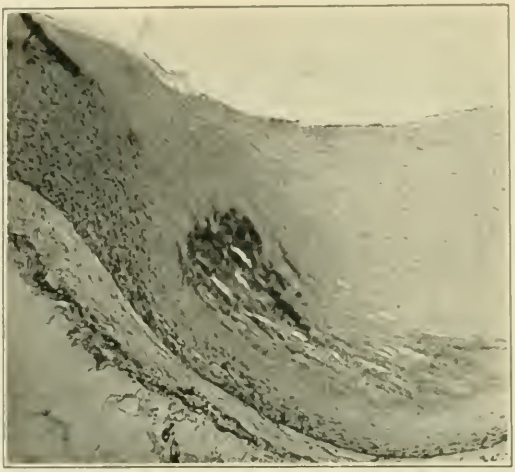

Fig. 18.—Radial artery of a blacksmith, aged seventy years (Case 52), a syphilitic, with chronic fibrous myocarditis, chronic mitral and tricuspid endocarditis and hypertrophy and dilatation of the heart, who died of pneumonia (hematoxylin and eosin). Extensive sclerotic plaque of intima with calcification. Widespread sclerosis of media which opposite the plaque is much thinned and stretched. Clinically this was a thickened vessel.

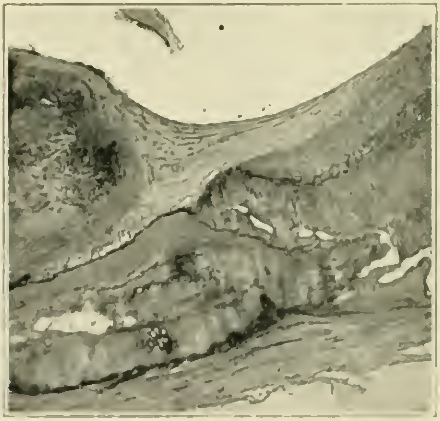

Fig. 19.—Radial artery of a farmer, aged eighty-three years (Case 1), with chronic nephritis and hypertrophied heart, who died of bronchopneumonia (hematoxylin and eosin). Extensive calcification of the intima and media. Clinically this vessel was hard and nodular.

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[3] Wesen und Entwickelung der Arteriosklerose, Wiesbaden, 1903, S.

[4] Sitzungsberichte der Gesell. zur Beförderung der gesammten Naturwissenschaften zu Marburg, 1905, Nr. 8, 117.

[5] Op. cit.

the first decade. With growth and the associated increase in pressure the artery strengthens itself—in the *intima* by the separation of one or more fresh strands of elastica from the inner surface of the fenestrated memberane, and the appearance about these of a few connective-tissue cells and longitudinal muscle fibres; in the *media* and *adventitia* by a gradual hypertrophy and hyperplasia. After full growth has been reached, at the end of the second decade, there is little change in the thickness of the adventitia. The intima, however, tends to become slightly thicker from the development of more elastic tissue and smooth muscle fibres, while the depth of the muscular media shows a slight increase. Gradually, however, during the third and fourth decades, especially in individuals subjected to heavy physical strain, there appear areas in the intima in which there is a distinct, connective-tissue thickening; the regular elastic strands which are separated from the interna are replaced by numerous, finer, more irregular fibrils, while on the outer side of the elastica, a delicate layer of connective tissue also appears. Opposite these areas the elastica interna is less deeply undulating and seems somewhat stretched, while connective tissue begins to appear between the muscle fibres of the media. *In other words, the strain has begun to tell upon the vessel wall, and the yielding tube fortifies itself by the connective-tissue thickening of the intima and to a lesser extent of the media.* As Aschoff has well expressed it, the artery of the child and that of the young adult with its increased amount of elastic tissue has a low, elastic resistance but a high limit of elasticity. Gradually, with the wear and tear of age or as the result of unusual and excessive strain, the elastic resistance becomes unequal to the burden imposed upon it and the limit of elasticity tends to be passed. The giving vessel then strengthens itself with a substance which has a much higher elastic resistance, but a lower limit of elasticity, namely, connective tissue.

But these changes are not marked until the fifth decade when the arte y begins to assume an essentially different appearance. The vessel wall, unable to stand the strain, tends to stretch, and an additional support is offered by the development of firm, connective tissue in the intima, together with an increase in that upon the medial side of the interna and, in lesser degree, throughout the media. The vessel is here usually felt as a firm tube.

Finally, in these sclerotic vessels, degenerative changes set in, which are somewhat different from those seen in the larger arteries, consisting as they do, of local areas of coagulation necrosis with calcification, especially marked in the deep layers of the connective tissue thickenings of the intima and in the muscle fibres of the media, particularly opposite these points. These changes may, as has been pointed out by Mönckeberg, go on to actual bone formation. Although, in one instance, there was an apparent atheromatous softening in the sclerotic intima of a man, aged seventy years, in not one of our cases did we see the superficial proliferation so common with fatty changes in the aorta.

When these facts are considered it becomes evident that it may often be difficult to draw a sharp line between the normal and the pathological radial artery. That which is normal at 50, would be pathological at 30 years. A connective-tissue thickening of the intima is not to be regarded as pathological in old age.

Regressive changes, necrosis, calcification, and atheroma which, as Jores has pointed out, occur especially, if not only, in a sclerotic intima, must be regarded as pathological.

It is a striking fact that in not one of our cases did we see calcification of the media unaccompanied by similar changes in the intima.

Is the normal radial artery ever palpable? To this question there can, of course, be but one answer. In the great majority of these cases, after the age of twenty years, the radial artery was palpable. Recognizing the fact, however, that if one rolls the artery carefully against the bone the normal vessel is often palpable, we divided our cases, clinically, into those which were not palpable or palpable and not regarded as thickened, and in which the vessel appeared to be distinctly thickened. When we compared our clinical notes with our pathological observations the result was extremely interesting. In the first and second decades none of the arteries were considered as thickened; in the third and fourth decades 77 per cent. were regarded as essentially normal, but in the fifth decade, at exactly that period when the connective-tissue thickening of the intima becomes especially marked, it appears that 63 per cent. of the arteries were regarded, clinically, as thickened vessels.

The arteries which were regarded clinically as thickened showed, in the majority of cases, anatomical thickening of the intima.

The beaded, goose-necked artery corresponded in practically every case to calcification of the intima or of intima and media.

It was our impression that the great frequency of the palpability of the arteries might be due in part to the fact that the majority of our patients were colored. But an analysis of our tables seems to show that, while the frequency of palpable arteries is indisputably somewhat greater among the colored, the difference is by no means as marked as we had fancied that it would be. The observations of one of us (Thayer) show clearly that the percentage of palpability of the radials in this group of hospital patients was much higher than that in healthy individuals in higher walks of life.

A study of the individual cases shows that, as a rule, the thickened vessel becomes evident to the palpating hand. There are, of course, striking individual exceptions, especially in very fat people. The palpability of the vessel depends apparently on the general thickening of the coats, especially, however, upon the changes in the intima which are progressive throughout life. In cases of aortic insufficiency and in chronic nephritis in which measurements show a considerable thickening of the media as well, this doubtless plays a part in the palpability of the vessel, but according to our measurements and tables the part is subordinate. It is a striking fact, moreover, that the thickening of the intima and the media go usually hand in hand. An analysis of our tables shows that in those cases in which heart hypertrophy was found, the media and intima were almost uniformly thicker than the average, and selecting from these cases those in which aortic insufficiency or chronic nephritis was present, the thickening of both coats appeared, on the whole, yet, more marked, but *it was rather striking that the change was more noticeable on the whole in the intima than in the media.* The same observations are true of those cases in which especially high pressure was noted *intra vitam.*

Comparison of the changes in the radial with those in the mesenteric artery and aorta.

The structure of the *mesenteric artery* is in some ways different from that of the radial.

In *gross*, the appearances are much the same but on a larger scale. The same transverse striations are visible in the opened vessel of the young adult, and the irregular, longitudinal lines are present in the more thickened artery. The *elastica interna* is a much thicker band. The *media* contains much more elastic tissue which is arranged as a well-marked meshwork. In areas throughout the muscular coat there are conspicuous aggregations of these fibres. The *adventitia* is very much thicker and contains more elastic tissue than that in the radial artery; this tends to have a longitudinal course (Figs. 19 and 20).

The changes in the intima and media are in the main identical with those in the radial. The same elastic, muscular intima develops early in life and the same connective-tissue thickening in the later decades. Here, also, the change from elastic muscular to connective-tissue thickening begins to be specially noticeable in the fifth decade.

Two points, however, are striking in the mesenteric arteries which have been examined, namely: (1) Calcification is apparently much less frequent than in the radials. (2) In several cases plaques were seen with fatty softening of the deeper layers of the intima and superficial proliferation—a picture which we have never seen in the radial.

In the *aorta*, as pointed out by Jores, the elastic-muscular intima

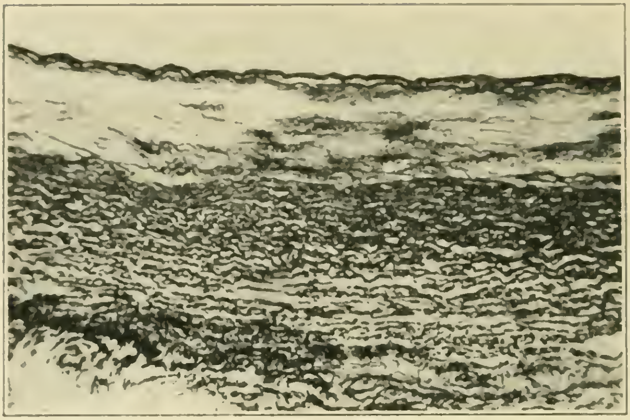

Fig. 20.— Mesenteric artery of a child, aged five years (Case 61), who died of bronchopneumonia following measles (Weigert's elastic tissue stain). The specimen shows the different arrangement of the elastic tissue from that seen in the radial artery. A second strand of elastic tissue has been separated from the surface of the interna.

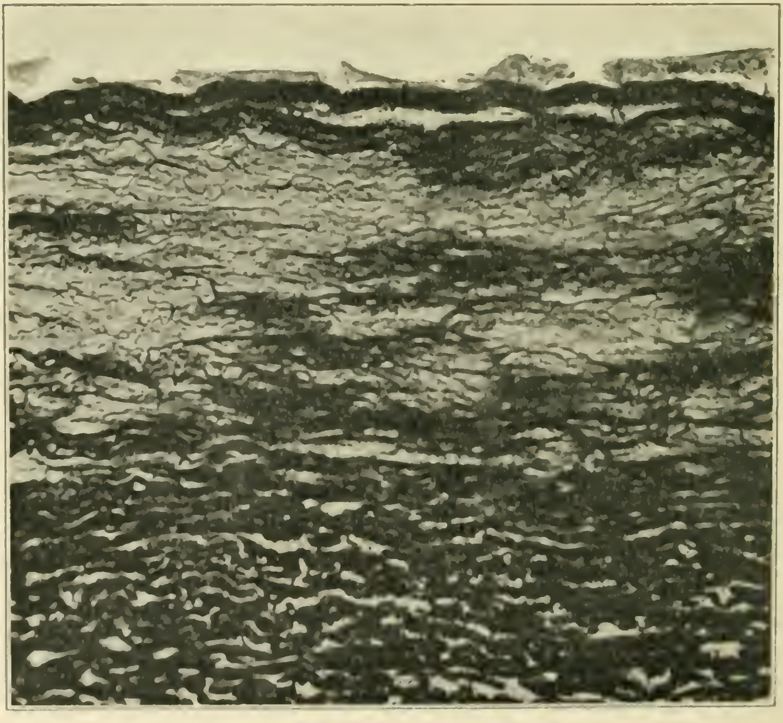

Fig. 21.—Radial artery of a child, aged five years (Case 61), who died of bronchopneumonia following measles (Weigert's elastic tissue stain). Shows the same conditions as Fig. 20, but under a higher power.

thickens progressively with age. This is beautifully borne out by Fabyan's measurements. Indeed, our tables, showing the thickness of the intima of the radial and the mesenteric arteries and the aorta, side by side, show a striking, progressive increase in the depth of the intima from birth to old age in all vessels, with the onset of connective-tissue sclerosis especially notable in the fifth decade. The disposition of the elastica in the aortic intima is much less regular than in the other vessels. The regressive changes, commoner in the thicker intimas, are particularly prone to be in the form of necrosis with fatty change and softening (atheroma) associated with active proliferation of the intima on the surface of the plaque.

In the aorta and mesenteric artery likewise over-strain and high pressure appear to result in changes analogous and more or less parallel to those in the radials. There are exceptions in individual cases which are sometimes striking. But, on the whole, when one finds an undue thickening of the intima in the radial, analogous changes are usually found in the mesenteric artery and aorta.

What inferences, then, are we justified in drawing from the presence of a thickened radial artery?

In old age a thickened radial artery represents conditions which are normal and to be expected, not only in peripheral, but in central vessels.

An unduly thickened radial at an earlier age may mean one of two things:

1. The vessel has been subjected to unusual and exceptional strain, or

2. It is a vessel which, from inherent weakness or other individual circumstances, has been unable to cope with conditions which might ordinarily be regarded as normal.

In either case the result has been the same; the artery has been obliged to fortify itself by progressive thickening of its walls, especially by a connective-tissue sclerosis of the intima and media.

As a rule, although there are striking individual exceptions, when the thickening of a radial artery is *unduly* marked, similar changes occur in the intima of the mesenteric artery and aorta. The early sclerosis of the intima, associated as it commonly is with like alterations in the muscular coat, constitutes a predisposition to those regressive changes (calcification, atheroma) which may be dangerous in other parts of the body. The distribution of such changes is irregular and too often not to be determined *intra vitam*, but the unduly palpable radial artery indicates a strong possibility of their existence in some part of the body at least, and may thus reasonably be regarded as a signal of danger.

THE SIGNIFICANCE OF TUBERCLE BACILLI IN THE FECES.

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The detection of tubercle bacilli in solid or well-formed stools of several patients whose histories were vague and in whom the clinical diagnosis was far from clear, led me to take up this subject for research. The idea was actually to see how prevalent is the occurrence of acid-fast bacilli in the feces, by studying the feces of others than those suffering from known tuberculous infection. With this end in view during the last two years, 612 cases were collected from the wards of the Philadelphia General Hospital. Together with this number there were stools of 60 cases of diagnosticated tuberculous infection, making in all 672. The stools were obtained from patients with croupous pneumonia, typhoid fever, erysipelas, diarrhœa, surgical and nervous disorders, and from individuals who were apparently healthy; in fact, from patients in all wards of the institution, no matter what the clinical diagnosis. Of these stools 137 were solid, 297 were semisolid, and 178 were fluid. The tubercle bacillus was found in 120 cases, or 19.6 per cent.; in solid stools 28 times; in semisolid stools 40 times, and in fluid stools 52 times. In the 60 cases of diagnosticated tuberculosis, the organism was demonstrable in all, no matter what the consistency of the feces.

It has been observed by some writers that acid-fast bacilli have been found in the feces of those suffering from enteric fever, and that it is quite common for them to be found in all specimens of feces even in health; that this latter assertion is absolutely erroneous will be shown by the results of the studies here recorded. The presence of the tubercle bacillus in the feces may be a process of excretion; wherefore a few experimental studies regarding the excretion of bacteria in general may be quoted.

Newmann[1] and Karlinski[2] observed Bacillus typhosus in the urine in 35 out of 112 cases examined. Wyssokowitsch[3] in 22 experiments employed fourteen bacterial species; only twice were the species introduced into the blood, and yet they were found in the content of the intestine. On each occasion there were macroscopic hemorrhages into the serosa and mucosa. He concludes that the passage of bacteria into the excreta occurs only when the blood containing them escapes through some breach of continuity in the excretory membrane due to inflammatory or mechanical injury. Dobroklouski[4] in Cornil's laboratory found that the bacillus of avian tuberculosis, when administered with food, infected guinea-pigs by penetrating the healthy intestinal mucosa.

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[1] Berl. klin. Woch, 1890, p. 229

[2] Prager med. Woch, 1890, p. 231.

[3] Zeits. f. Hyg., 1886, vol. i, p. 3, quoted by Sherrington.

[4] Quoted by Sherrington, Jour. Path. and Bact., 1893, p. 258.

As corroborative evidence, numerous investigators have performed experiments by feeding, by subcutaneous and intravenous inoculations, and proved that the organisms pierce and reach the mucous membrane without any evident lesion being present.

Emmerich and Buchner,[5] after injecting Bacillus neapolitanus into the blood, found that the organism escaped in large numbers through the intestinal wall. In one experiment no pathological change in the intestinal wall was observed, but in almost all there was blood in the contents of the intestine and hemorrhages in the mucosa. Sherrington,[6] in concluding his article on the escape of bacteria with the secretions, says the evidence is against believing that when the transit of bacteria across the secreting membrane occurs the membrane is still normal in condition, although at the same time it may not be ruptured or pervious to red blood cells.

The fact that the escape of the bacteria tends to occur not immediately upon the introduction of them wholesale into the circulation, but in the late stages of the communicated disease, suggests that the healthy secreting membranes are not pervious to bacteria, and that only after soluble poisons produced by the infection have had time to act upon them do the membranes become pervious to the germs.

Babes[7] concludes, from experimental work upon glanders, that the bacillus can penetrate uninjured mucous membranes. At this point may be mentioned the well-known experiments of Ravenel, who, shortly after having fed animals tubercle bacilli, killed them and found the bacilli in the thoracic duct, though no lesions or abrasions of the mucous membrane of the gut were evident.

As another instance of the penetration of mucous membranes by pathogenic organisms, typhoid bacilluria may be mentioned. This condition was studied at some length by Koujojeff,[8] by Charrin and Ruffer, Ruffer, Schweiger, Blachstein, Corrado, Pernice and Scagliosi, Cornil, and others, quoted by Sherrington.[9] They have proved, by experiments upon animals with various bacteria, that it is quite common for bacteria to be found in the bile or urine after intravenous or subcutaneous inoculation or even after feeding alone.

Griffith[10] has shown that when tubercle bacilli are injected into a cow the milk may be infective without disease of the udder.

I inoculated rabbits and guinea-pigs subcutaneously with a homogenized culture of attenuated human tubercle bacilli. Upon the fourth day I detected tubercle bacilli in their feces. After seven weeks the animals were killed. The guinea-pig showed neither a local nor visceral lesion, not even intestinal lesions, while the rabbit, though no visceral lesions were found, had a caseous mass at the site of inoculation and in the right inguinal region. A second series of guinea-pigs and rabbits was inoculated in the same manner and with the same organism. I again found tubercle bacilli in their feces at the end of the fourth day. These animals were killed at the end of the sixth week and at autopsy no intestinal, visceral, or glandular lesions were present in the guinea-pig, but the rabbit showed a large, apparently caseous mass in the right inguinal region in which I was unable to find tubercle bacilli.

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[5] Arch. Hyg., vol. xi, p. 357.

[6] Jour. Path. and Bact., 1893, p. 276

[7] Comptes-rend. Paris, 1888.

[8] Central. f. Bact. u. Parasit., 1889, vol. vi, p. 672.

[9] Loc. cit.

[10] Royal Commission on Tuberculosis, Brit. Med. Jour., 1907, ii, 211.

Examination of a number of specimens of feces from guinea-pigs and rabbits in apparent health failed to reveal any acid-fast bacilli.

In well-defined instances of pulmonary tuberculosis it is the rule to find tubercle bacilli in the feces. If a case comes under the observation of the clinician which is not at all clear, but presents a clinical picture resembling malaria, enteric fever, or acute miliary tuberculosis, the finding of tubercle bacilli in the feces will determine the diagnosis. In those suffering from chronic diarrhœa, with no other appreciable symptoms, pulmonary or otherwise, the tubercle bacillus is at fault in most cases. These cases have frequently come to autopsy, some showing intestinal ulceration and others showing no ulceration.

Instances of ascites, the exact nature of which was unknown, were diagnosticated positively as tuberculous by the finding of the tubercle bacillus in the feces. These cases were further proved by surgical procedures, that is, abdominal operation. As a well-marked intestinal tuberculous ulcer is appreciable from the appearance of the serous coat of the gut, in one case thus operated upon, no ulcers were present.

That tubercle bacilli are in the feces, irrespective of a pulmonary or an intestinal lesion is proved by the fact that I found them in cases of general glandular involvement, meningitis, hip-joint disease, and Pott's disease of the spine, the latter condition being in a boy aged five years.

In acute miliary tuberculosis, diagnosticated or not clinically, the bacillus was present in the feces in all cases. The result of these studies suggests the intestinal mode of infection in tuberculosis in general. It is my intention merely to quote a few observations supporting the theory of the intestinal infection in tuberculosis, as these studies strengthen that theory. This theory of infection is gaining ground daily and the experiments of Schroeder and Cotton,[11] of Ravenel, and of Vallee who practically confirmed Ravenel's feeding experiments by working upon calves, of von Behring, and Calmette, and Guerin's work upon goats, demonstrate fully that the intestine is by far the most common path of infection and that aërial infection is uncommon.

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[11] Bull. No. 86, Bureau of Animal Industry of Department of Agriculture.

In a number of autopsies in which the mesenteric and other glands were studied bacteriologically, it was found that over 40 per cent. showing no tuberculous lesions in any part of the body were tuberculously infective. It was also found that in all cases of active tuberculosis and in almost all cases of inactive tuberculosis, the mesenteric glands were tuberculously infective.[12] Supporting von Behring's theory that the most frequent method is through the intestinal wall, Guthrie found over 22 per cent. of infections through the intestinal tract, Heller nearly 40 per cent. of primary intestinal affections, and Still over 23 per cent.

To explain the presence of the tubercle bacillus in the feces of man is not easy. I believe that the bacillus enters the human economy through ingestion (water?), either in infancy or maturity. At first the number of bacilli is not large, and they find their way to the blood and lymph stream. During their transit some are discharged through the feces and others through the urine. The circulation of these organisms through the lymph and blood continues indefinitely, and the patient actually suffers from a toxemia which may be so severe as to set up chronic diarrhœa, or cause vague symptoms characteristic of no one disease.

As the organisms multiply in the body and as the toxemia and irritation progress, a point of least resistance is somewhere established; the tubercle bacillus lodges and sets up the disease, with its distinct pathological features. It is interesting to note that in cases of healing tuberculosis, or actually arrested or healed cases, the tubercle bacillus is rarely if ever found in the feces. This is also true of the occurrence of tubercle bacilli in the sputum. In some of these cases recorded, the sputum has been examined at least six and as many as twelve times negatively, and subsequent examination of the feces was also negative.

Passler,[13] in considering the diagnosis of pyretic conditions, as septicemia and typhoid fever, mentions the probability of acute intestinal tuberculosis being the cause of the malady, irrespective of tuberculosis of any other organ. During the evolution of intestinal tuberculosis, he suggests that the infection by pyogenic organisms will perform the same role as they do in ulcerative pulmonary tuberculosis, and asserts that when we are dealing with a marked pyretic condition, which has lasted for several weeks without any particular and definite signs to indicate typhoid fever or septicemia, we must always think of an acute intestinal tuberculosis. He concludes by saying that as an intestinal infection may occur without any manifestations clinically the feces should be examined for the presence of tubercle bacilli. He cites only 2 cases.

Wood[14] states that tubercle bacilli are found in the feces of persons suffering from tuberculosis (pulmonary), because in the majority of cases the bacilli are swallowed together with small masses of sputum. Sahli[15] mentions that in intestinal tuberculosis tubercle bacilli are found in the feces and are therefore of diagnostic importance. "The stools may, however, contain these bacilli even though there is no intestinal tuberculosis (if the patients swallow their sputum). Searching the stools has even been recommended for the diagnosis of lung tuberculosis in cases of irresponsible persons who swallow the sputum. Previous treatment with dilute potassium hydroxide or digestive enzymes is often successful and may be serviceable in the examination of mucopurulent particles of the movement which have been isolated from the mass of feces. We do not know whether, under certain conditions, decomposition will destroy tubercle bacilli in the intestine. At any rate, we cannot always demonstrate tubercle bacilli in the stools, even when there is undoubted intestinal tuberculosis. Perhaps this is on account of the dilution of the content of tubercle bacilli by the abundant particles of food. Tubercle bacilli are most readily found in the purulent or bloody pieces of diarrhœal stools. As tubercle bacilli in the feces may be due to swallowed sputum, we can diagnosticate intestinal tuberculosis if bacilli are found in the feces only, when at the same time, attacks of diarrhœa occur with pus and blood in the stool. The tubercle bacillus must be carefully distinguished from the smegma bacillus, which is said to occur at the anal orifice and might have become mixed with the feces."

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[12] Rosenberger, A Study of the Mesenteric Glands in their Relations to Tuberculosis, AMER. JOUR. MED. SCIENCES, July, 1905.

[13] Münch med. Woch., October 23, 1906.

[14] Chemical and Microscopical Diagnosis, 1905.

Lichtheim[16] says that the presence of tubercle bacilli in the stools is the exception rather than the rule in persons suffering from pulmonary tuberculosis. He further asks the question whether the presence of these organisms in the feces means intestinal ulceration or merely that the patient has swallowed them with his sputum. In control observations he showed this not to be the case, as he claimed it was only exceptional to find the bacilli, since they were very difficult to find and then only a very few were present.

In January, 1897, Shaw[17] mentions the finding of tubercle bacilli in the feces of a patient exhibiting no tuberculous lesion of the intestinal tract. The lungs, however, showed isolated tubercles and areas of bronchopneumonia.

Emerson,[18] in remarking upon the occurrence of tubercle bacilli in the feces, says that it must always be borne in mind that the organisms may be swallowed, especially in children in whom the diagnosis of pulmonary tuberculosis has been made; "but this is rather a remote possibility in the case of a careful adult."

Boston[19] recommends collecting a "small portion of the purulent or mucoid material from the feces, smear it thinly on a slide," and then stain as for tubercle bacilli in the sputum. "Tubercle bacilli when found in the feces point conclusively to the existence of tuberculous ulceration of the intestines."

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[15] Diagnostic Methods, 1905.

[16] Fortschritte d. Med., January, 1883.

[17] Jour. Amer. Med. Assoc., March 20, 1897, p. 551

[18] Clinical Diagnosis, 1906, p. 390.

[19] Ibid., 1904, p. 380.

Simon[20] claims that when tubercle bacilli are present in the feces, it indicates intestinal ulceration, providing they are observed upon repeated examinations and there are clinical symptoms pointing to the bowels as the seat of the disease; otherwise, they may be referable to swallowed sputum.

Technique. If the feces to be examined was fluid or semisolid, a small quantity from any part of the stool was taken and spread on a slide, dried, and stained. When the feces were solid, a small amount of sterile distilled water was put upon the slide and a small mass of fecal matter added, mixed thoroughly, spread, dried, and stained. Not one of the specimens was centrifugalized.

In staining the preparation, carbol-fuchsin was applied for fifteen minutes in the cold, the excess drained off, and Pappenheim's solution poured on the preparation. This was allowed to act for two or three minutes, washed with water, and if the specimen was of a uniform blue color it was dried and examined in cedar oil. If the preparation was not uniformly blue, Pappenheim's solution was applied and reapplied until the smear was blue. By observing this technique carefully, no mistake can happen regarding the diagnosis of the tubercle bacillus, as this organism and spores of other bacilli are the only bodies retaining the carbol-fuchsin stain. All other bacteria and cellular elements are stained blue. Great care must be taken lest some artefact be mistaken for the tubercle bacillus, such as a minute scratch in the glass, a small crystal, or the periphery of a cell. The organisms, as a rule, are comparatively few, in cases not plainly diagnosticated as tuberculous, but in well-marked cases of pulmonary or intestinal tuberculosis they are comparatively abundant. The finding of the tubercle bacillus in a spread is not always easy of accomplishment; it has frequently taken me at least an hour and sometimes as long as two hours to find three or four bacilli.

Direct searching through solid stools is less promising than in fluid stools. Nevertheless, tubercle bacilli may quite frequently be demonstrated in solid movements, if, as Hamberger[21] recommends, we mix a piece of feces the size of a pea with a few centimeters of water, then centrifuge gently to remove the coarser pieces, dilute the supernatant cloudy fluid with a double volume of alcohol, centrifuge once more, and then after drying examine the remaining precipitate which will consist almost exclusively of bacteria. Personally I have never found this procedure necessary.

Park[22] recommends searching in the feces for any purulent or mucous particles, and if none are found the larger masses are removed, the rest diluted and centrifugalized, and stained by the ordinary methods.

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[20] Clinical Diagnosis, fifth edition, 1904, p. 328.

[21] Quoted by Sahli.

[22] Pathogenic Microörganisms, 1905, p. 312.

Page[23] mixes a small mass of feces in 1.5 c.c. distilled water, adds 54 c.c. of a mixture of equal parts of alcohol and ether, centrifugalizes ten minutes, makes a smear of the sediment, fixes it to the slide with albumen, and stains as usual.

If the assertion that the tubercle bacilli found in the feces result from swallowing sputum or the presence of intestinal tuberculosis is true, the examination of the feces is useless, as no further knowledge is gained.

It is a well-known fact to students of tuberculosis that a persistent diarrhœa is present for a very long time, and yet the autopsy shows no ulcerations in any part of the intestinal canal. Therefore, it is by no means certain, if we find tubercle bacilli in the feces of those suffering from tuberculous enteritis, that ulcerative lesions are present. But, suppose that acid-fast bacilli are present in the feces of a person not suffering from any appreciable lesion of tuberculosis, what then is the significance of such a finding?

I believe from the studies made of this number of cases, both from a clinical and pathological standpoint, that if an acid-fast bacillus is present in the feces of any individual, and this organism resembles morphologically and tinctorially the tubercle bacillus, tuberculosis of some part of the body exists. I do not mean pulmonary tuberculosis, but tuberculosis of the intestines, liver, lymph nodes, peritoneum, or any viscus.

A *resume* of some of the cases that came to autopsy, with general remarks concerning some of the patients who did not succumb to the disease, is of great interest and very instructive, and is appended. In almost two-thirds of the cases I was fortunate in being able to follow the clinical findings with those at autopsy. A few of the patients were removed from the institution in a precarious condition, and although a number of them died no autopsy was permitted.

Case I.—Clinical diagnosis, cirrhosis of liver with ascites. There was no expectoration. Three days before death delirium set in, suggesting meningitis. At autopsy miliary tuberculosis of lungs and tuberculosis of the peritoneum; no intestinal ulcerations.

Case II.—Clinical diagnosis, chronic pleurisy. Eleven examinations of sputum were negative. At autopsy pleura was one-eighth of an inch in thickness over right lung and the same lung showed miliary tuberculosis. No intestinal ulcerations.

Case III.—Clinical diagnosis, acute miliary tuberculosis. The sputum was examined on nine occasions with negative results. At autopsy miliary tuberculosis of lungs and spleen. No intestinal ulcerations.

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[23] Quoted by Emerson.

Case IV.—Clinical diagnosis, acute miliary tuberculosis. At least six examinations were negative for the tubercle bacillus. At autopsy there was found acute miliary tuberculosis of all the viscera; no intestinal ulcerations.

Case V.—Clinical diagnosis, pleurisy and tuberculosis of hip. Sputum on a number of occasions was negative for the tubercle bacillus. At autopsy there was acute miliary tuberculosis of all viscera; no intestinal ulcerations. (Three weeks after finding the tubercle bacillus in the feces, it was found in the sputum.)

Case VI.—Clinical diagnosis, typhoid fever. No expectoration, and there were four negative Widal reactions. At autopsy six small, irregular, atypical ulcers; long axis transverse to bowel; one of these ulcers had perforated the gut. Studied bacteriologically, these ulcers showed few tubercle bacilli.

Case VII.—Clinical diagnosis, tuberculosis of the hip. No autopsy.

Case VIII.—Clinical diagnosis, acute miliary tuberculosis. Acute miliary tuberculosis of all viscera was found at autopsy, but no intestinal ulcerations.

Case IX.—Croupous pneumonia and pleurisy. No tubercle bacilli found in the sputum. Crisis occurred, but there is still an irregular temperature.

Case X.—Clinical diagnosis, chronic diarrhœa. No cough; no expectoration. No autopsy.

Case XI.—Clinical diagnosis, chronic diarrhœa. After finding tubercle bacilli in the feces, slight impairment of resonance was noted in the right apical region.

Case XII.—Clinical diagnosis, chronic diarrhœa and cirrhosis of the liver. No expectoration. Tuberculous ulcers were found in the gut at autopsy.

Case XIII.—Clinical diagnosis, repeated attacks of pleurisy. No expectoration. No autopsy.

Case XIV.—Clinical diagnosis, pleurisy with effusion. Sputum negative on four occasions for the tubercle bacillus.

Case XV.—Clinical diagnosis, chronic diarrhœa. No expectoration. Typical tuberculous ulcers of the gut were found at autopsy.

Case XVI.—Clinical diagnosis, erysipelas and diarrhœa. No expectoration. Slight pulmonary lesions were found; no intestinal ulcers.

Case XVII.—Clinical diagnosis, septicemia, pelvic abscess, typhoid. Acute miliary tuberculosis of all viscera; no intestinal ulcers.

Case XVIII.—Clinical diagnosis, malignant endocarditis. No expectoration. Acute miliary tuberculosis of all viscera; no intestinal ulcers.

Case XIX.—Clinical diagnosis, chronic diarrhœa. At autopsy, intestinal ulcers were found.

Case XX.—Clinical diagnosis, alcoholism. No expectoration. Recent and old lesions were present in the lungs; no intestinal ulcers.

Case XXI.—Clinical diagnosis, tuberculous peritonitis. At autopsy tuberculous peritonitis and intestinal ulcers were found.

Case XXII.—Clinical diagnosis, pleurisy. Tuberculosis of both pleuræ, of mesenteric glands and spleen; no pulmonary or intestinal lesions found.

Case XXIII.—Clinical diagnosis, chronic diarrhœa. Tuberculous ulcers present.

Case XXIV.—No clinical diagnosis made. At autopsy general miliary tuberculosis was found, but no intestinal lesions.

Case XXV.—Clinical diagnosis, enteritis. This case was a child two years old and treated for seven months for gastro-enteritis; there was no cough, no expectoration; the cervical glands were enlarged.

Case XXVI.—Clinical diagnosis, tuberculous peritonitis. Patient had cavities in both lungs, yet no sputum could be collected.

Case XXVII.—Clinical diagnosis, Pott's disease. This case was a child five years old; no pulmonary lesions demonstrable. No autopsy.

Case XXVIII.—Clinical diagnosis, typhoid fever and pneumonia. No tubercle bacilli were demonstrable in the sputum on several different occasions, and during illness two negative Widal tests resulted, and blood showed a leukocytosis of 17,000. At autopsy acute miliary tuberculosis of both lungs, liver, and spleen, with few intestinal ulcers.

Case XXIX.—Clinical diagnosis not made. At autopsy, general miliary tuberculosis; no intestinal ulcers.

Case XXX.—Clinical diagnosis, tuberculous peritonitis. This case was operated upon and the diagnosis confirmed.

Case XXXI.—Clinical diagnosis, probable carcinoma of the stomach. Pernicious vomiting, chronic diarrhœa, marked cachexia, and emaciation were among the cardinal symptoms. No autopsy.

Case XXXII.—Clinical diagnosis, typhoid fever. Child six years old, running a persistent irregular temperature; no pulmonary symptoms, and three Widal tests were negative.

Case XXXIII.—Clinical diagnosis, secondary anemia. Besides the tubercle bacillus being found in the feces, the ova of Tricocephalus dispar and Ascaris lumbricoides were present. The malarial parasite was also seen in the blood. (As this was an immigration case, he was immediately deported.)

Case XXXIV.—Clinical diagnosis, probable tuberculosis of the liver. Bacilli were found on two occasions in the feces. No autopsy.

Case XXXV.—Clinical diagnosis, not made. Sputum examination negative on three occasions. Caseous tuberculosis of the bronchial and mediastinal glands; miliary tuberculosis of the kidney, liver, spleen, and intestines was found at autopsy.

Case XXXVI.—Clinical diagnosis, chronic pleurisy. No tubercle bacilli were found in the sputum. At autopsy adhesive pleurisy on both sides was found, together with a few miliary tubercles in the lung. There was no intestinal ulceration.

Case XXXVII.—Clinical diagnosis, erysipelas. No tubercle bacilli were demonstrable in the first three examinations of the sputum, though a fourth examination was positive. (Three days after tubercle bacilli were found in the feces, pulmonary hemorrhage took place, and the bacillus was found at this time.)

Case XXXVIII.—Clinical diagnosis, interrupted recovery from typhoid fever. No autopsy.

Case XXXIX.—Clinical diagnosis, typhoid fever. Acute miliary tuberculosis of a general character was found at autopsy; no intestinal ulcerations.

Case XL.—Clinical diagnosis, chronic diarrhœa. Tuberculosis of the intestines and tuberculosis of the mesenteric glands were present at autopsy.

Case XLI.—Clinical diagnosis, acute miliary tuberculosis. No tubercle bacilli were found in the sputum. At autopsy acute miliary tuberculosis of all the viscera was found, but no intestinal ulcers.

Case XLII.—Clinical diagnosis, chrônic diarrhœa and jaundice. *X*-rays showed gallstone. Tuberculosis of the retroperitoneal glands was observed at autopsy, but no gallstone.

Case XLIII.—Clinical diagnosis, pneumonia, no crisis, and running an irregular temperature; no tubercle bacilli could be found in the sputum. Healed tuberculosis of the lungs, with tuberculous ulcers in the gut, were found at autopsy.

Case XLIV.—Clinical diagnosis, typhoid fever. No expectoration. At autopsy acute miliary tuberculosis of a general character was found.

Case XLV.—Clinical diagnosis, carcinoma of the liver. At autopsy, cancer of the lung and liver were found, together with tuberculosis of the mesenteric glands.

Case XLVI.—Clinical diagnosis, locomotor ataxia with empyema. One liter of pus was found in the pleural cavity at autopsy. There was one positive and two negative examinations of the sputum for tubercle bacilli.

Case XLVII.—Clinical diagnosis, general glandular enlargement. Spreads from an inguinal gland removed during life showed tubercle bacilli, and spreads from mesenteric gland removed at autopsy also contained tubercle bacilli. At autopsy there was no pulmonary tuberculosis, but a purulent peritonitis, with general enlargement of all lymphatic structures. No intestinal ulcers.

Case XLVIII.—Clinical diagnosis, typhoid fever. At autopsy, tuberculous ulcers were observed; no pulmonary lesions.

Case XLIX.—Clinical diagnosis, tuberculous peritonitis. Operation disclosed fibrinopurulent peritonitis. Numerous miliary tubercles on visceral and parietal layers of the peritoneum were observed, but no intestinal ulcerations could be made out.

Case L.—Clinical diagnosis, malaria. Patient had chills, fever, and sweats, occurring almost daily for ten days. Examination of the blood on several occasions failed to demonstrate the malarial parasite.

Case LI.—Clinical diagnosis, meningitis. Child, eighteen months old; no pulmonary symptoms. Tubercle bacilli were demonstrable in the spinal fluid about the same time they were found in the feces.

Case LII.—Clinical diagnosis, cervical lymphadenitis. Upon the second examination and after a very careful and prolonged search a few tubercle bacilli were found in the feces.

Case LIII.—Clinical diagnosis, cutaneous tuberculosis (lupus). Although tubercle bacilli were found in the feces, no pulmonary lesions could be made out.

Case LIV.—Clinical diagnosis, diarrhœa alternating with constipation. No autopsy.

Case LV.—Clinical diagnosis, gunshot wound of the chest penetrating the lung. On five occasions the sputum was examined for tubercle bacilli, but with negative results. At autopsy a large cavity was found which was surrounded by gangrenous tissue, and on the margin, upon histological examination, tubercles were found which showed few tubercle bacilli.

Case LVI.—Clinical diagnosis, tumor of testicle, probably tuberculous. The organ was removed and histologically presented a typical picture of tuberculosis. No pulmonary lesions were evident. It might also be mentioned that tubercle bacilli were found in sections of the organ and also in spreads before fixation.

Case LVII.—This case was one in which a large, ulcerating mass was present in each groin, following the inguinal glands. The condition had persisted for two years. Tubercle bacilli were found in the feces, though the condition had been diagnosticated as sarcomatous, specific, and tuberculous. Sections of the masses studied histologically showed typical tubercles and giant cells and tubercle bacilli.

Besides these cases just cited there were 9 cases of chronic diarrhœa, some of which, upon autopsy, showed tuberculous ulcers and others did not. In a few of these latter cases slight pulmonary involvement could be made out, while in the greater number no pulmonary lesions were demonstrable.

Conclusions. 1. No acid-fast bacillus other than the tubercle bacillus was found in the feces.

2. The presence of the tubercle bacillus in the feces means that active tuberculosis exists somewhere in the body.

3. In acute miliary tuberculosis the bacillus is always present in the feces.

4. In all cases of chronic diarrhœa and in cases of general glandular involvement, the feces should be examined for tubercle bacilli.
5. Finding tubercle bacilli in the feces does not mean intestinal ulceration in all cases.
6. In arrested or healed pulmonary tuberculosis no tubercle bacilli are found in the sputum or the feces.
7. The feces should be studied for tubercle bacilli as a part of the routine examination, especially in suggestive cases and when no expectoration can be obtained.

I wish to extend my thanks to the various resident pathologists at the Philadelphia General Hospital for their valuable coöperation.

ACUTE INTUSSUSCEPTION IN THE ADULT.

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Intussusception, although far more common in children, occurs with sufficient frequency in the adult to warrant a careful study of the causes, pathology, and results. One-third of all cases of intestinal obstruction are due to intussusception. In 1000 cases of acute intestinal obstruction reported by C. L. Gibson,[1] 187 were due to intussusception, 121 to volvulus, 354 to hernia, 42 to Meckel's diverticulum, 40 to gallstones, 34 to openings, 16 to foreign bodies, 20 to miscellaneous causes. Leichtenstern[2] found 442 cases of intussusception in a collection of 1152 of intestinal obstruction, excluding congenital obstructions, stenosis of the rectum, and the various forms of hernia. The exact proportion of cases in children and adults is about 65 to 35. It is the most common cause of intestinal obstruction in children, and one of the most uncommon in adults. Treves gives the following table of frequency by age for acute intussusception: Before the age of eleven years, 53 per cent.; between eleven and twenty years, 12 per cent.; between twenty-one and forty years, 20 per cent.; between forty-one and sixty years, 11 per cent.; beyond sixty years, 4 per cent. or less. Sixty-five per cent. of all cases occur before the age of twenty, and 53 per cent. before eleven years. Twenty-five per cent. occur during the first year of life. In chronic intussusception the percentage differs: 50 per cent. of this class of cases occurs between twenty-one and forty, and only 25 per cent. under eleven years.

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[1] Annals of Surgery, 1900, p. 487.

[2] Vierteljahreschrift f. d. prakt. Heilk., 1873-74.

There are three anatomical varieties of intussusception: enteric, colic, and ileocolic. Of those occurring in the region of the ileocecal valve there are three forms: ileocecal, ileocolic, and a combination of these, called by Leichtenstern, iliaco-ileo-colica. The most common form of invagination is the ileocecal, the most uncommon being the ileocolic; the other varieties coming between these two in the order of enteric and colic.

Invaginations of the small bowel rarely occur in the upper portion of the intestinal tract, although a case is reported of the presence of duodenum in the invaginated portion of an ileocecal intussusception.[3] In one of our cases the invagination was of the jejunum, about three feet from the duodenal junction. Most enteric invaginations occur in the lower portion of the jejunum, next in frequency being the ileum, in about the proportion of 4 to 1.[4] In our first case the amount of gut invaginated was about three inches long, and this corresponds with the general experience of observers, that enteric invaginations usually involve relatively small portions of gut. The shortness of the invaginated portion does not interfere with the severity of the symptoms, or with a fatal termination if the condition be unrecognized and unrelieved.

Any portion of the large bowel may be involved in the colic variety of intussusception: the ascending into the transverse, the transverse into the descending, the descending into the sigmoid, or the sigmoid into the rectum. Colic intussusceptions are necessarily short, as the large bowel is fixed by its short mesentery.

Ileocecal intussusception is the commonest form. The ileocecal valve forms the apex of the invaginated portion of bowel. The ileum does not pass through the valve, but pushes the valve ahead of it. This form may attain great size. Cases are on record in which the valve traversed the entire length of the colon and presented itself at the anus, and even protruded through the sphincter. In the ileocolic variety the terminal portion of the ileum invaginates, passing through the ileocecal valve. The valve and cecum remain at their normal position and only later are carried into the colon. The third variety, iliaco-ileo-colica, is a combination of the ileocecal and the ileocolic, and is really an advanced stage of the ileocolic, in which the ileum is first protruded through the valve or down to it, and the cecum is then forced with the invaginated ileum into the colon.

The cause of all intussusception of the bowel is irregular muscular action, the muscles in the wall of bowel acting normally in one portion, while in an adjoining portion muscular action is abnormal, due to one of several causes. This abnormality may be a limited but severe muscular action, or a total muscular paralysis—spasmodic invagination and paralytic invagination. The experiments of Nothnagel have placed the pathology of intussusception on a fairly

[3] Dr. Delipun, Path. Soc. Trans.

[4] Treves Int. Obstruction, p. 144.

firm basis. He demonstrated the two forms, spasmodic and paralytic, in rabbits. In the spasmodic form the circular muscular fibers were made to contract, narrowing the lumen of the gut, while the adjacent bowel remained normal in size and activity. When he stimulated the bowel to the stomach side of the contracted portion, nothing happened. When, however, the bowel to the lower side was stimulated, the longitudinal fibers, by their contraction, caused the normal bowel to turn in on itself causing a progressive descending invagination, in which the contracted portion of bowel did not take place. The same thing occurred when the bowel was totally paralyzed by crushing. When an intussusception increases in length it does so at the expense of the sheath. The method of increase of the invaginated portion of the ileum in the ileocolic variety is different from the other varieties of intussusception; here the ileum protrudes through the ileocecal valve and meeting no resistance in the large cecum, grows by the protrusion of more and more ileum, and not at the expense of the sheath, the limit being reached by the length of the mesentery. Clinically, the spasmodic variety is the only one met with, and the vast majority are descending invaginations. Retrograde or ascending invaginations occur, but are of short duration and a very small portion of bowel is invaginated. As a rule, they reduce themselves and are, therefore, not often recognized although often suspected as the cause of transitory intestinal colic. Double and triple intussusception also occur but rarely. A recent reported case will be recited later.

Irregular muscular action of the intestinal muscularis is the requisite for intussusception. The causes which give rise to the irregular action are, ptomaïne poisoning, enteric fever, traumatism, growths of the bowel, benign and malignant. Ptomaïne poisoning, causing, as it does, violent action of the muscle of the bowel by the local irritation, and this action being irregularly distributed through the bowel, gives rise to the majority of cases of intussusception. The vast majority of cases of intussusception occur in children in the milk-feeding period. Impure milk is an extremely common cause of intestinal poisoning, hence we can reason from cause to effect. This frequency in children of intestinal colic due to infection, plus the increased mobility of the large bowel, is corroborative of this opinion. In the adult intussusception from ptomaïne poisoning is less common. The following case is in point:

G. M., agent, aged twenty-seven years, was referred to the German Hospital, August 3, 1905, by Dr. George Y. MacCracken. On August 1 he had eaten deviled crab and watermelon; he said the crab was bad. Several hours later he had a severe abdominal pain, colicky in character, and associated with vomiting. His abdomen was scaphoid, but there was no tenderness or rigidity. Morphine did not relieve the pain. Castor oil did not produce a stool. On the following day his abdomen was still scaphoid, with general tenderness, but no rigidity and no tumor could be made out; he was still vomiting and there was no bowel movement. He was admitted on August 3, with marked symptoms of intestinal obstruction. No tumor could be made out on account of the abdominal distention. Pulse 108; temperature normal. He had an anxious expression and his appearance was of one suffering from profound toxemia. Dr. MacCracken had diagnosticated ileocecal intussusception. His abdomen was rapidly opened and the diagnosis proved to be correct, except that it was an ileocolic invagination. It was impossible to reduce the invagination, which involved a large segment of the ileum, probably eighteen to twenty inches, and so an ileocolotomy with a Murphy's button was hastily performed. The man's condition forbade any further procedure. He lived but a few hours and died of shock and exhaustion.

We believe the exciting cause of this case was ptomaïne poisoning from decayed crab meat.

That intussusception occurs during the course of enteric fever is proved by the following recorded cases, which have come to notice since the practice of opening the abdomen for perforation or suspected perforation has come into vogue. Lieutenant-Colonel Jennings,[5] R. A. M. C., reports 2 cases occurring during enteric fever, giving rise to symptoms similar to perforation. The true condition was not discovered in either case until the necropsy was performed.

Ash[6] reports two cases, one in which six inches of ileum had slipped into the cecum. Operation six hours after the onset of symptoms. The patient, a soldier, aged twenty-five years, on the ninth day of a relapse was suddenly seized with acute abdominal pain which passed away only to return in an hour with vomiting and marked rigidity, but without distention. The pain was referred to the umbilicus and epigastric region. Under anesthesia a mass could be felt in the right iliac fossa which was dull to percussion. The patient was operated upon, the intussusception was reduced and the patient made an uninterrupted recovery. The second case reported by the same author, was discovered at postmortem; the third case was operated on by one of us (Ross), November 25, 1903, at the German Hospital.

Dr. J. C. Wilson speaks of invagination of the bowel occurring during the course of enteric fever, but relates no cases. This statement was probably founded on postmortem findings. Hart and Ashhurst[7] speak of having seen it occur during an attack of ambulatory typhoid. B. L. Bryant and J. S. Bragg[8] report a case, occurring in a man, aged twenty-three years. Ordinary course of typhoid fever. In fourth week sudden abdominal pain, localized in right iliac fossa. Pain intense, skin moist, small bowel movement. Temperature, 102°; pulse, 120; respiration, 24. Abdomen became very tender, most intense one inch above McBurney's point. One hour after attack, pulse 145, and weak. Abdomen not distended. Patient quiet. Diagnosis, perforation. Operation: three intussusceptions both ways: the first a short distance from ileocecal junction was eight inches long and easily reduced; the second, five inches long, a short distance from the first, was also easily reduced; and a third, three inches long, also in the ileum, was discovered. Patient died five hours after operation.

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[5] British Medical Journal, 1902, i, 1083

[6] British Medical Journal, May 3, 1902.

[7] Annals of Surgery, January, 1904

[8] Med. Record, November 18, 1905, p. 818

Two cases have been operated on at the German Hospital. The first has been reported by one of us (Ross[9]), and the other was operated on by Dr. Deaver.

Case I.—W. B., a male, aged seventeen years, in a severe attack of typhoid fever (with hemorrhages), was awakened on the twenty-sixth day with violent abdominal pain. He was thought to have perforation of the intestine, but operation disclosed an intussusception three inches from the junction of the duodenum and the jejunum. This was readily reduced, and although he had later hemorrhages, he made a good recovery. (Details of the case history may be found in the original report.)

I do not believe that there is a special pathological cause for intussusception under these circumstances, except it be the lack of muscular tone due to inflammatory infiltration of the bowel wall in localized areas, permitting a greater degree of distention in different portions of the bowel.

Case II.—Miss J. S., aged nineteen years; single; a wireworker, was admitted February 27, 1906, on the nineteenth day of the disease, and was discharged on May 20, 1906. Diagnosis: enteric fever. Complications: hemorrhages, intussusception. Operation: reduction. Relapse: cholecystitis. Sequel: neuritis, with foot drop.

The family history is negative.

Previous History. The patient had measles, pertussis, varicella, and pneumonia (at six years). Menstruation established at fourteen years; regular but painful.

For several months she had felt indisposed and low-spirited. For two weeks before admission she had been confined to bed with high fever (according to her statement). Vomited once; had slight abdominal pain and some diarrhœa. No epistaxis; no cough, but considerable expectoration.

The physical examination was negative, except that the spleen was enlarged to percussion. No rose spots were found. On March 2, the Widal was negative, and on the 13th it was positive.

On the twenty-first day of the disease, three days after admission, and on the two subsequent days the patient had hemorrhages. There were eight bloody stools, amounting to 2040 c.c. The temperature dropped 8° F., from the evening of the twenty-first day to the morning of the twenty-third day of the disease. There was a rise again until the temperature reached 101.8° F. on the twenty-seventh day. Then followed a gradual decline until, on the evening of the thirty-fifth day, the record was 98.4° F. From this time there was another rise to 103.4° on the thirty-ninth day. On the fortieth day, at 2 A.M., the patient had some abdominal pain and vomited a yellow fluid with some mucus. At 11 A.M. she passed a small, liquid stool containing bloody mucus. At 2 P.M. the pain in the abdomen grew worse. At 5 P.M. another stool containing shreds of bloody mucus was passed. Later there were several more stools with streaks of blood and mucus. There was considerable tenesmus. During the day patient vomited six times. The vomitus changed from a yellow at 2 A.M. to light green at 5 A.M.; dark green at 11 P.M.; brown at 4.30 A.M. on the forty-first day; dark brown with fecal odor at 11.30 A.M. On the forty-first day, at 8 A.M., the abdominal pain became very severe, peristalsis was greatly increased. The leukocyte count was 11,000. A diagnosis of intussusception was made and the patient was transferred to the surgical department for operation.

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[9] Surgical Complications of Typhoid Fever. Amer. Jour. Med. Sciences, 1905, cxxx, 115.

Operation. Dr. Deaver. Chloroform. Vertical incision 9 cm. long in the outer margin of the right rectus muscle. Upon opening the peritoneum, the cecum was found to be collapsed. The ileum was very much injected and distended. A large ulcer was found in the ileum near the cecal junction and an intussusception at this point. The cecum was the intussuscipiens, and 25 cm. of the ileum the intussusceptum. The ileum was withdrawn without difficulty. The patient reacted well from the operation. From the forty-first to the forty-sixth day her temperature ranged from 101.4° to 103.4° F. On the forty-sixth day she was transferred to the medical department with a relapse. The temperature was 103° F. She was weak and restless, pulse rapid and feeble, the respirations frequent.

On the fiftieth day the stitches were removed. Wound in fine condition. Hemorrhage from left nostril. Bowels quite free. Pulse rate 152. On the fifty-ninth day the gall-bladder became painful, tender, and palpable. Some cough. On the sixty-sixth day she developed a neuritis in the right leg with subsequent foot-drop. On the seventy-third day the temperature reached normal and remained normal until the patient's discharge from the hospital on the one hundred and second day. She still suffered with partial foot-drop, but otherwise was in good condition.

For 2 cases illustrating traumatic intussusception and that due to malignant growths we are indebted to Dr. Francis T. Stewart, surgeon to the Germantown Hospital. The notes as given by Dr. Stewart are as follows:

Traumatic Intussusception. N. G., aged thirty-six years, was struck just above the crest of the left ilium by a heavy steel beam. Shortly afterward he was admitted to the Germantown Hospital in the most profound shock. At the end of twenty hours his temperature had risen to normal and the pulse had fallen to 110. He had vomited twice and had passed twelve ounces of bloody urine. There was great pain all over the abdomen, a large hematoma in the left loin, and intense rigidity of the abdominal muscles. Liver dulness was decreased by three fingers' breadth, and no dulness could be detected in the flanks. The abdomen was opened in the middle line below the umbilicus and a large extraperitoneal extravasation of blood found extending from the bladder, whose walls were infiltrated with blood, to the left kidney which was normal to palpation. The abdominal muscles on the left side were torn from the left iliac crest. The peritoneal cavity was opened and found to be clean; there was no visceral rupture. In numerous places the small intestine was tightly contracted, the areas involved varying greatly in extent, so that in certain regions the intestine seemed to be ligatured, while in others it resembled a piece of tape. In one place the contracted intestine had passed into the relaxed segment below for a distance of two inches. The intussusception was reduced, the peritoneal cavity closed, and the extraperitoneal tissue drained. The patient died four hours later of shock.

Intussusception due to Malignant Growth. C. H., aged forty years, had sarcoma of the iris removed about nine months ago and later the eyeball for recurrence. Now has general melanotic sarcomatosis. He was admitted on April 7, 1906, with severe abdominal pain and non-fecal vomiting for twenty-four hours, elongated mass in right side of abdomen becoming harder with colic, no blood or mucus from rectum, no distention; bowel movements have been growing smaller for the past two or three days. Incision on right side, intussusception about one foot long of ileum into ileum, apex of the intussusception entering colon and reaching hepatic flexure, the apex of the intussusception being formed by a tumor about one-half inch in diameter in the bowel wall. This, as well as several other tumors, showed marked umbilication. Intussusception reduced with difficulty. Recovery from operation uneventful.

The diagnosis of acute intussusception in children is less difficult than in the adult. In children it is the most common cause of intestinal obstruction, and children are very much less liable to inflammatory affection of the peritoneum and abdominal organs, thus offering less obscurity to the trouble. It must be remembered that all acute intussusceptions do not progress to obstructive conditions. It is probable that a very large percentage of attacks of acute severe abdominal pains are due to temporary, short invaginations which quickly relieve themselves by unfolding, the incarceration being short-lived.

The onset of the attack is usually sudden. Treves thinks that 75 per cent. of acute cases have a sudden onset. There is usually evidence of some one of the predisposing causes preceding the seizure.

This is more commonly noted in adults, as we have the benefit of the individual's ability to state the circumstances relating to the attack. The mode of onset is influenced by the portion of the bowel involved. In the ileocolic the commencement is always sudden, while that of the large bowel is most often gradual. In patients who give no apparent cause for an attack, stating that they were in perfect health up to the time of onset, it is well to investigate carefully the food ingested during the preceding twenty-four or forty-eight hours, for we believe that a satisfactory explanation, namely, ptomaïne poisoning, will be found to account for the 52 per cent. in which no evident exciting cause has been offered.[10]

The most persistent and also the first symptom is pain. It may reach its maximum intensity when the invagination occurs and then moderate, although it is usually more gradual, becoming more and more severe until the parts become fixed or gangrenous, when the pain diminishes or ceases altogether. This tendency to diminish in intensity is significant and should always lead to a suspicion of the trouble. The character of the pain is colicky and intermittent at first, becoming more constant as the obstruction progresses. This is in accordance with the rule that intermittent pain almost invariably indicates partial obstruction, while in complete obstruction the pain is constant and more severe and without marked exacerbations. In the incomplete obstruction the pain may not completely disappear, but there are distinct exacerbations. The pains are at first general and ill defined, becoming localized later. Invaginations of the small bowel give rise to more intense pain than those of the large bowel, due to the greater muscular development of the former. The location of the pain is indefinite until the invagination becomes fixed, when it localizes to the area of bowel involved.

Vomiting is most common in the acute intussusception. It is not so severe as in other forms of intestinal obstruction, and does not appear as early, nor is there the same tendency to stercoraceous vomiting. It is likely to occur in intervals and gives great temporary relief. This temporary relief is a characteristic feature of intussusception. As a rule the higher the invagination the more severe the vomiting.

Rigidity of the abdominal muscles is not met with in intussusception in the early stage, coming on only after peritonitis has set in. Distention is also absent in the majority of cases, only making its appearance late in the attack when the obstruction is complete, or marked peritonitis supervenes. In cases in which marked diarrhœa is an early symptom the belly is apt to be scaphoid. Tenderness is a late symptom and indicates peritonitis. In the early stages pressure offers distinct relief.

The presence of a tumor, which in children and in chronic intussusception is of great diagnostic value, is not met with so often in acute cases in the adult. Leichenstein found a tumor present in about 50 per cent. of all cases in all ages. It is most frequently met with in the cecal and ileocolic varieties. In none of our cases was a tumor demonstrable. When present it may be discovered through the abdominal walls or in the rectum. It varies in size with the size of the intussusception, but never gives an idea of the actual amount of bowel invaginated. It is usually hard and resisting. It is also usually more prominent during attacks of pain and sometimes has a distinct range of motion.

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[10] Treves' table of Intestinal Obstruction, p. 178.

The temperature, as a rule, remains normal or slightly above normal, except in cases associated with shock. Collapse, however, is far less common than in obstruction due to bands. It is far more common in young infants and is then rarely fatal. Tenesmus in acute and subacute attacks is a severe, early, and constant symptom. The nearer the invagination is to the anus the severer the tenesmus. It may or may not be associated with blood or blood-stained mucus. Thirst in intestinal obstruction depends upon the degree of vomiting, hence is less marked in intussusception than other forms of strangulation.

We believe that operation is the safest and most logical procedure in the treatment of acute intussusception. To have the best results early diagnosis and prompt action are essential. Any attempt to reduce a well-defined intussusception by the use of forced enemas is in our opinion losing valuable time.

PRIMARY CARCINOMA OF THE URINARY BLADDER.

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A review of the literature has failed to reveal a recent report of any considerable series of cases of primary carcinoma of the bladder thoroughly studied from both the clinical and the anatomical sides. Going back a few years there is no lack of material from which to draw, and it is from this source that most of our literature is derived. The marked discrepancies in the statistics and in the statements of the various authors would lead one to infer that most of the original literature will not bear a very close inspection, and this is to a certain extent borne out by the facts. Carcinoma of the bladder is a hidden disease. One cannot note its beginning and watch its progress from day to day. Furthermore, its location and malignant nature make its clinical manifestations peculiarly subject to confusion with those of complicating or neighboring diseases. Such being the case, it is very evident that statistics gleaned from the records of the clinician and the surgeon may be very inaccurate, and the inferences based thereon no safe guide. On the other hand, the conclusions of the pathologist and the microscopist, widely at variance with those of the practitioner, need comparison with the reports of the latter.

The above considerations have furnished me with the excuse for the report of the following series of 10 cases, the records and specimens of which are in the pathological laboratory of Rush Medical College. I wish to emphasize the fact that the report is based primarily upon postmortem examinations, supplemented in each case by a careful microscopic examination, and as far as possible, by the clinical data. In each instance the following questions were answered in the affirmative: (1) Is the tumor malignant? (2) Is it a carcinoma? (3) Is it primary in the bladder?

The ultimate standard of malignancy was that set by Ziegler; namely, infiltration of the bladder wall or the formation of metastases. Parenchymatous infiltration of the muscularis was demonstrable, microscopically, in every case; the epithelial character of the tumor was established by the histological appearance; and, without exception, we were able to exclude the possibility of prostatic or other extravesical origin.

Case I.—A man, aged thirty-nine years. The clinical history was not to be obtained. At the autopsy was found: "a condition of right pyonephrosis and left hydronephrosis; the left ureter is dilated to the size of a lead pencil. The bladder contains a grayish-brown, purulent fluid. The inner wall is studded with nodules, varying in size from that of a split pea to a walnut. The prostatic urethra is invaded and a tumor, situated on the trigone, obstructs the ureters." A study of microscopic sections showed the growths to be papillary carcinomas with extensive necrosis, and with finger-like projections extending down into the muscular wall.

Case II.[1]—J. S., male, aged forty-six years, a musician, was first admitted to the Presbyterian Hospital in the summer of 1901, complaining of pain and strangury on urination. At that time a diagnosis of cystitis was made. After his discharge the patient's symptoms increased in severity and he lost flesh. He developed an irregular fever with occasional chills. He returned to the hospital, and was operated on March 26, 1902, a suprapubic incision and perineal drainage being performed. Several small calculi were removed from the bladder, and a diagnosis of carcinoma was made from the scrapings. Two days later a blood examination showed a rather marked anemia with a color index of 0.6, and a leukocytosis of 18,600. The urine was acid until a few weeks before death, when it became alkaline. Death occurred April 30.

The postmortem examination showed a secondary involvement of the retroperitoneal, pelvic, and mesenteric lymph glands and growths in the lungs and liver. In addition there was an ascending cysto-uretero-pyelonephritis with dilatation of the ureters and renal pelves on both sides. The entire inner surface of the bladder wall, including the urethral and both ureteral orifices, was invaded by tumor tissue, which projected in large, necrotic masses and showed extensive ulceration. The anterior wall was adherent to a subperitoneal carcinomatous mass and was torn in separating the organ from the symphysis. A perforation appeared to have taken place at that point and to have been walled off by adhesions. Microscopic examination showed that the growth was what may be described as a pseudotubular carcinoma, the cells being, for the most part, arranged in solid cords and nests, but showing in places the appearance of imperfect tubules. The muscular wall was extensively infiltrated.

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[1] Reported in Trans. Chicago Path. Soc., 1902, v, 105

Case III.—H. K., male, aged forty-four years, was admitted to the Norwegian Tabitha Hospital. The patient gave a history of gonorrhœa at twenty-two years. This was closely followed by strictures, cystitis, and pyelonephritis. Two years later a urethrotomy was performed, after which he enjoyed good health until the spring of 1901. At that time urination became frequent and painful. In January, 1902, he fell on his right hip. Two months later a small, hard tumor developed in the reigon of the right sacro-iliac joint, the urinary symptoms in the meantime growing progressively worse. An operation was performed for the removal of this osteal tumor on May 5. The patient developed an irregular fever after the operation and died May 27, 1902. The urine was acid throughout and contained a large amount of albumin and numerous pus cells.

The autopsy revealed: "A carcinoma of the bladder with metastases in the right iliac bone, left eighth rib, right second and ninth ribs, liver, mesenteric, retroperitoneal, inguinal, and peribronchial lymph glands. Two of the affected ribs have become fractured. There is an ascending pyelonephrosis with dilatation of the right ureter and renal pelvis, and also a psoas abscess. The bladder contains a large amount of gravel glued together with mucus and adherent to the mucosa in the form of balls. The tumor, as a diffuse thickening of the bladder wall with projecting ridges 1 or 2 cm. in width at the base, occupies almost the entire lower one-third of the bladder and extends up on the right wall. Both ureteral orifices are involved in the tumor mass, the right being completely closed. The projecting point of one of the ridges partly blocks the internal urethral orifice. The prostate is smooth externally and of normal size. The tumor is of rather hard consistency and is not ulcerated." The microscope showed the muscularis to be extensively infiltrated. The sections presented the appearance of a papillary carcinoma, with cords of cells arranged in a glove-like, or arborescent manner.

Case IV.—O. C., male, aged forty-three years, a farmer by occupation, admitted to the Presbyterian Hospital, gave a history of gonorrhœa nineteen years before his present trouble began. He denied other illnesses. In November, 1901, his first symptoms developed: frequent urination accompanied by a burning sensation. His symptoms grew more severe, he lost weight, and at the time he entered the hospital was having occasional chills accompanied by an irregular fever. There was a slight pyuria and hematuria. The reaction of the urine was alkaline. A blood examination showed the red cells to be 3,912,000; the white cells, 8050; the hemoglobin, 55 per cent. A nephrotomy was performed. The patient survived this operation two weeks, dying November 23, 1902.

The postmortem findings were: "A primary carcinoma of the urinary bladder, with secondary involvement of the kidneys, adrenals, lungs, and left testicle. There is an ascending cysto-ureteropyelonephritis, and a right perinephric abscess. A stone is impacted in the lower portion of the right ureter; the proximal end of the ureter and the pelvis of the kidney are dilated. There is a slight stricture of the membranous urethra. The external urethral orifice opens at the junction of the glands with the corpus spongiosum." "The upper four-fifths of the bladder wall shows an extremely necrotic, ulcerated, greatly thickened, lining. Superficially the growth is nodular and fungoid. It extends to within 1 cm. of the urethral opening, while the ureters open just below the margin of the tumor mass." The prostate and seminal vesicles were not involved. Microscopically the tumor was found to be a typical papillary carcinoma which had very extensively infiltrated the bladder wall.

Case V.—H. M., male, aged seventy-two years, was admitted to the Norwegian Tabitha Hospital, to the service of Dr. Sandberg. The family history was negative and the patient denied venereal disease. He had been in good health up to October 15, 1902, when his first symptoms began: severe, lancinating pains in the back and abdomen, radiating to the right thigh and testicle. These continuing, he was admitted to the hospital on November 17. At that time his urine was acid, contained much albumin and numerous red cells. He had no fever. On November 22, about one-half of the bladder, comprising the greater part of the anterior wall, was resected. There was no fever subsequent to the operation, but persistent nausea and vomiting developed, and death supervened December 24.

At the postmortem a considerable necrosis of the bladder wall was found near the operation wound. There was a general, acute, fibrinohemorrhagic peritonitis, and an acute cystitis and urethritis. Metastatic tumors were found in the liver, lungs, and the right eighth rib, which had sustained a pathological fracture. The ureters and kidneys were practically normal. Quoting from the records, "The left lobe of the prostate is 4 by 2.5 cm. The cut surface is slightly gelatinous; the right lateral lobe answers the same description."

The microscope showed no indication of tumor formation in this organ. Sections of the bladder wall showed that it was deeply infiltrated by tumor cells, evidently those of a papillary carcinoma, of the rapidly-growing type and with a marked tendency to degenerative changes. The secondary tumors were similar to this in character.

Case VI.—P. J., male, aged thirty-eight years, was admitted to the Presbyterian Hospital to the service of Dr. Robison. No statement as to previous diseases was obtained. The patient was ill in May, 1902, with an affection diagnosticated as "malaria." His urine was bloody and there was frequent and painful micturation. The following winter the hematuria again appeared, and the patient became weak and emaciated. He entered the hospital, and the diagnosis of carcinoma of the bladder was made. Toward the close he developed gastro-intestinal "distress" and a cough. A blood examination showed: red cells, 3,880,000; white cells, 14,800; hemoglobin, 45 per cent.

Only the contents of the abdominal cavity were examined postmortem. Metastatic tumors were found in the liver, mesenteric, and retroperitoneal lymph glands. There was ascites and slight prostatic hypertrophy. A tumor, composed of flat, sessile, soft projecting masses, occupied the inner surface of the posterior wall of the bladder, beginning 3 cm. above the upper border of the prostate and extending to the highest point of the bladder. The tumor showed an extensively ulcerated area and was covered in part by a thick layer of gravel. On microscopic examination both the muscular wall and the serous covering of the bladder were found to be infiltrated with strands and clusters of tumor cells, the arrangement justifying the classification of papillary carcinoma.

Case VII.—C. E., male, aged sixty-four years, was admitted to the Presbyterian Hospital. This patient, a bricklayer, denied venereal infection, and said that he had used alcoholic drinks in moderation. He gave a history of excellent health up to April, 1903, when urination became difficult, frequent, and painful. On his entrance into the hospital July 16, 1903, his bladder was distended with bloody urine. Rectal examination revealed a prostate the size of a lemon. His temperature was 100.2°. A blood examination showed a red count of 4,670,000; leukocytes, 5200; and hemoglobin, 52 per cent. A prostatectomy was performed July 21. Fever and severe chills followed, and death ensued three days later.

Among the anatomical findings were, "Ulcerative carcinoma of the urinary bladder with secondary involvement of the pelvic and retroperitoneal lymph glands, including a gland at the bifurcation of the aorta." The tissues were all distended by gas generated by the Bacillus capsulatus aërogenes.

Quoting further from the autopsy records, "A large tumor mass measuring 14 by 10 cm., made up of a large number of projecting

Examined October 28, 1911. Full supination, pronation 165 degrees. Class I.

Case 19.—Juliette J., aged eight years. July 3, 1906. Children's Hospital. Greenstick, middle third. Treated in full supination. Examined August 2, 1906. Class I.

Case 20.—Carrie C., aged six years. August 2, 1906. Children's Hospital. Injury two weeks ago at Atlantic City, and dressed in mid-pronation. On admission today, fracture at junction of middle and lower thirds of right forearm; no union, fair position; but bowing of ulna to extensor surface. Treated in full supination. August 21, union good, no deformity; full supination and pronation. Examined August 28, 1906. Upper fragment of ulna displaced slightly posteriorly; functions perfect. Class II.

Case 21.—John S., aged twelve years. August 6, 1906. Children's Hospital. Left, junction of middle and lower thirds. Fell from tree; was stunned. Upper fragment of ulna projects beneath skin of flexor surface; both lower fragments displaced toward extensor and radial surfaces. Treated in full supination. August 10. Deformity persists; pad over upper fragment of ulna. August 14. Position better; growing firmer. August 21. Position fair; bones firm. Examined September 5, 1906. Little deformity, no disability; supination and pronation complete. Class II.

Case 22.—William G., aged seven years. November 3, 1906. Episcopal Hospital. Left, greenstick, middle third. Dressed in full supination. Then patient visited another dispensary, and when splint applied there fell off of itself, he returned to Episcopal Hospital, November 19, with marked dorsal bowing of both bones. Bones were re-fractured, deformity reduced, and forearm dressed in full supination. Examined December 10, 1906. No visible deformity, but a little callus palpable over radius. Full supination and pronation. Class I.

Case 23.—Albert S., aged eleven years. November 15, 1906. Episcopal Hospital. Right, complete of radius, greenstick of ulna. Dressed in full supination. Examined December 12, 1906. Class I.

Case 24.—Hugh F., aged fourteen years. November 21, 1906. Episcopal Hospital. Left, above wrist. Full supination. Examined October 28, 1911. Supination complete, pronation to 180 degrees. Class I.

Case 25.—Andrew M., aged fourteen years. December 22, 1906. Episcopal Hospital. Left; compound comminuted fracture of both bones of left forearm with compound comminuted fracture of left humerus. (Reported as Case VI in paper on "Multiple Fractures," in *Annals of Surgery*, 1907, ii, 263.) Treated in ward for nine days, then in dispensary. Forearm dressed in full supination. Fig. 6 is from a photograph taken three months after the accident. Examined October 28, 1911. Supination and pronation complete. No deformity. Figs. 7 and 8 are from skiagraphs made in January, 1912. Class I.

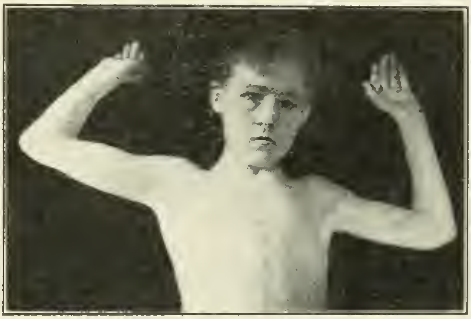

Fig. 6.—Case XXV. Compound comminuted fractures of both bones of left forearm, with compound comminuted fracture of left humerus. No deformity. Perfect function. See Figs. 7 and 8.

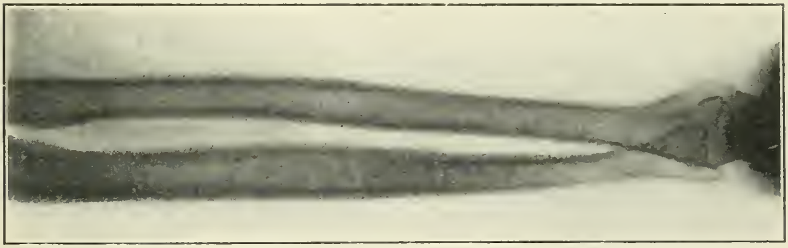

Fig. 7.—Case XXV. Lateral view of forearm five years after compound comminuted fracture of both bones.

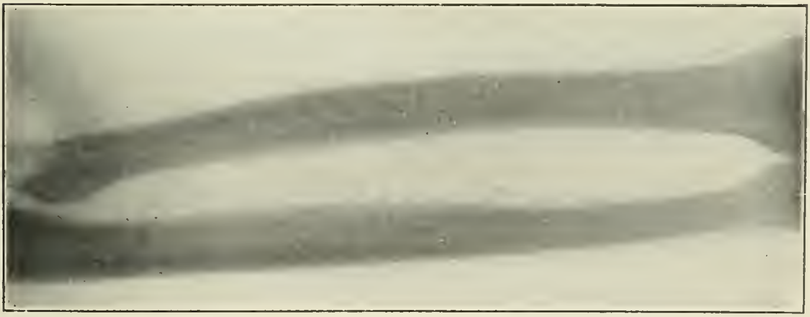

Fig. 8.—Case XXV. Antero-posterior view of forearm, five years after compound comminuted fracture of both bones.

Case 26.—Henry D. E., aged fifty-seven years. November 26, 1906. Episcopal Hospital. Left. Compound comminuted fracture of both bones of left forearm, with comminuted fracture of left humerus. (Reported as Case V, in paper on "Multiple Fractures," in *Annals of Surgery*, 1907, ii, 263.) Treated with

There is a condition of double pyonephrosis. Both ureters are markedly distended, measuring, when laid open, from 3 to 5 cm. in circumference. The right ureter contains a stone. On the right side is an accessory ureter extending from the pelvis of the kidney to a point on the ureter 7 cm. above its vesical orifice. Its lumen is patent throughout. The pelves of both kidneys are much dilated and the kidney substance diminished in thickness. In the left kidney several cavities extend from the pelvis into the cortex to within a few millimeters of the surface." There were no metastases. On microscopic examination the tumor proved to be a papillary carcinoma made up of large cuboid cells with vesicular nuclei and a rather scanty stroma.

A good example of the contradictory statements in the literature is met with in regard to the prevailing seat of the tumors. Barling,[2] in an analysis of 260 vesical neoplasms, found two-thirds springing from the base. Albarran[3] divides the bladder into three parts, the lower being below the level of the ureteral orifices. He affirms that most tumors originate in the middle third, and found only 2 out of 88 springing from the trigone; admitting, however, that of tumors originating elsewhere one-third extend to the trigone. On the other hand, Clado,[4] who observed 250 cases of malignant tumor, asserts that their seat of election is the base of the bladder and that extension nearly always takes place in an upward direction, involvement of the rectum, vagina, or prostate rarely taking place, while extension through the muscular wall and intestinal adhesions, consequent on an upward growth, is not at all uncommon.

In our series it could be pretty definitely determined that the seat of origin was in the trigone in three instances. In 1 case (II) it is uncertain whether the tumor had its start in the trigone or in the anterior wall; probably the latter. Three of the tumors showed greatest involvement of the upper part of the bladder while the remainder affected the middle zone more extensively. The urethra was directly involved in the tumor mass in two instances, while in 2 there was partial obstruction by projecting portions of the neoplasms. One or both ureteral orifices were directly implicated in 3 cases, while in 4 others there was partial obstruction by pressure of the tumor or nodules. One tumor (Case VII) had invaded both seminal vesicles, as was demonstrated both by external examination and by serial sections. Of such involvement Clado had not seen an example. None of the growths had extended to the prostate or rectum, and only one had caused perforation into the peritoneal cavity.

As to the frequency of extension to the glands and as to the formation of further metastases there is a wide divergence of opinion. Clado states that of 250 cases of malignant tumor observed in his clinical experience, there were only 4 instances of glandular involvement when the tumor was well localized in the bladder. He also quotes Fere, who in 145 cases found glandular involvement in only 10. Pasteau[5] asserts that the iliac glands are enlarged in 43 per cent. of all sessile and 85 per cent. of all infiltrating tumors, but that metastasis gets but little further than this. Keyes,[6] quoting this statement, concludes that "a vesical tumor (carcinoma or sarcoma) remains confirmed to the bladder for years and rarely gives rise to metastases of any importance."

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[2] International Clinics. Philadelphia, 1894, 4th series, i, 231.

[3] Les tumeurs de la vessie. Paris, 1891.

[4] Traité des tumeurs de la vessie. Paris, 1895.

On the other hand, Albarran, in 16 observations, found glandular involvement 11 times, and Tuffier[7] in 15 autopsies found 3 instances of propogation to neighboring organs and 11 of glandular infection and generalization. Bland Sutton[8] testifies to the frequency of involvement of the pelvic lymph glands and of general dissemination.

An analysis of these and other conflicting statements seems to show that, generally speaking, clinicians hold to the view that vesical carcinomas tend to remain localized and form few metastases, although pathologists incline to a contrary opinion. Several factors may be responsible for this discrepancy: The nature of the lymphatic system of the bladder is such that a malignant tumor would naturally tend to remain localized in the earlier stages, which are more likely to fall under the observation of the surgeon than the pathologist. According to Poirier and Charpe, lymphatic vessels probably do not exist in the mucous membrane of the bladder, but the network of origin is intramuscular, from which the efferents lead into a second network between the muscularis and peritoneum or perivesical fascia. The neoplasms are said to originate most frequently in the deeper layers of the epithelium, and the direction of growth is at first naturally along the line of least resistance, toward the inner surface of the bladder. Another factor for this discrepancy is that without a postmortem examination there is a strong probability that metastases, especially in internal organs, will be overlooked. This statement might appear entirely superfluous, did not one meet with so many examples in the literature of attempts to draw conclusions from clinical observations alone. Another factor is the ease with which a benign may be mistaken for a malignant tumor. There are reports of considerable series of operations for the extirpation of "carcinoma of the bladder" with little or no reference to microscopic examinations.

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[5] Etat du systéme lymphatique dans les maladies de la vessie et de la prostate. Paris, 1898, Pp. 46, 52.

[6] The Surgical Diseases of the Genito-urinary Organs. New York, 1899, 259.

[7] Traité de Chirurgie. Paris, 1892, vii.

[8] Tumors, Innocent and Malignant. Philadelphia, 1893.

The following statement by Lincoln Davis[9] is apropos in this connection: "It is generally conceded that of two tumors which are macroscopically and microscopically indistinguishable, the bladder wall may show infiltration in one and not in the other." Finally, there is abundant evidence that benign tumors may undergo malignant transformation, and it might happen that the observer of the final stages had no knowledge of the long period that the tumor had manifested no signs of malignancy.

In 3 cases of our series no metastases or extravesical involvement was found; in 1 they are doubtful, none being described. In the other 6 cases metastases were present in various organs, as follows: four times each in the retroperitoneal glands and lungs; twice in bones, and once each in the peribronchial glands, inguinal glands, adrenal gland, kidney, and testicle.

The occurrence of metastases in the bones in 2 cases might lead to the suspicion of an error in diagnosis, especially as there is but little reference in the literature to such occurrences. However, the possibility of the tumors being sarcomas or prostatic carcinomas was excluded positively. The secondary tumors in the bones in these 2 cases entitle carcinoma of the bladder to a place in clinical medicine and surgery with the prostatic, adrenal, mammary, thyroid, and uterine tumors.[10]

A point deserving of special emphasis is the mode of death. In 7 of our cases this was due to ascending infection of the urinary tract. In each of the 7 cases there was demonstrable blocking of one or both ureters; in 3 by direct involvement and in 4 by pressure. The ureters in several instances were so dilated and their walls so hypertrophied that they resembled more the small intestine than the ureter.

Reference has already been made to the difficulty of diagnosis in primary carcinoma of the bladder. Probably in many cases the condition is not ascertained until postmortem examination. In our series the correct diagnosis was made in 2 cases from the symptoms and physical examination. In 2 other instances it was made after cystotomy for complications, while in 6 the primary tumor was unrecognized clinically.

The reasons for such mistakes, aside from the inherent difficulty of separating symptomatically the various affections of the bladder and contiguous structures, are two-fold. The first is the character of the complications which are especially prone to obscure the diagnosis in the later stages. The other is the character of the early symptoms. In the literature on this subject one meets with the oft-reiterated and much emphasized statements, that painless, copious, repeated, apparently causeless hematuria is a very early symptom; in the majority of cases the first observed by the patient, and that its character is not far from being pathognomonic. Yet in only 3 of these 10 cases was hematuria, sufficient to be noticed by the patient, an early symptom. One other patient observed blood in the urine in considerable amounts, but some time after the onset of pain and strangury. In the 5 remaining cases whose clinical history was obtainable hematuria was late, and only slight or microscopic in quantity. It would seem that there is no clinical symptom or combination of symptoms by which we can affirm or deny the presence of a vesical carcinoma, and that the only basis for a positive diagnosis is offered us by the cystoscope and the microscope.

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[9] Tumors of Urinary Bladder. Annals of Surgery, 1906, xliii, 556-591.

[10] See report by Yoder, Description of an Osteoplastic Metastatic Carcinoma of the Sternum Following a Primary Carcinoma of the Uterus. Med. News, December 6, 1902.

Conclusions. (1) Intelligent deductions and trustworthy statistics are to be derived only from careful and thorough study of a large number of cases from both the clinical and the anatomical sides. (2) The most frequent seat of carcinoma in the bladder and the direction of extension are still debatable questions. (3) Metastasis is much more frequent than is conceded by many clinicians. (4) A malignant growth in the bones may occur from a primary carcinoma of the urinary bladder. (5) The most frequent cause of death in vesical carcinoma is a secondary infection of the ascending urinary tract. (6) The diagnosis is always difficult, especially after the onset of complications.

INCOMPLETE AND COMPLETE HYPOTHYROIDŒA OR MYXŒDEMA.

By Robert L. Pitfield, M.D.,
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Deficient secretion of the thyroid, according to the degree of subactivity and age of the subject, may cause at least three distinct clinical entities. Deficiency of secretion varying in amount from nearly normal to virtually none may produce mild nervous symptoms of a vague nature, or various grades of *myxœdema, idiocy,* or *cretinism:* just as an excess of secretion, or hyperthyroidœa varying in degree produces either tachycardia or profound intoxication with severe derangement of the whole body, or even death. Somewhere, then, between the normal secretion and almost total cessation of secretion, producing marked and complete myxœdema, there is a condition of subsecretion which brings about what is known in medical parlance as incomplete hypothyroidœa or "*myxœdeme fruste.*" Cases of this nature are described in detail by Murray,[1] Hertoghe,[2] Ewald,[3] Richardson,[4] Moffitt,[5] and Howard,[6] and they have been recognized as belonging to a definite disease group, because they do not tend to progress into a condition of complete myxœdema.

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[1] British Med. Jour., 1898; Twentieth Century Practice of Medicine.

[2] Nouv. Iconographe de la Saltpêtrière, 1899.

[3] Die Deutsche Klinik.

[4] The Thyroid and Parathyroid Glands, 1905.

[5] Jour. Amer. Med. Assoc., vol. xlv, No. 12.

Ibid., vol. xlviii, Nos. 14 and 16.

This disease has many things in common with the complete form. It is more frequent in women and among those in the fourth and fifth decade, especially in those who have borne many children, or it may not occur spontaneously, and follow partial thyroidectomy. There is always some structural change in the thyroid as in complete myxœdema, and there can be no doubt as to the causal relationship between thyroid fibrosis and atrophy, and hypothyroidœa.

The symptom complex is as follows: The heart is weak and often dilated. The skin is dry and scaly, and there are curious but typical pads of fat above the clavicle. Very often there are violent joint pains, paresthesias and neuralgias. Associated with these there is a marked asthenia, subnormal temperature, great dyspnœa, and a characteristic *giving way of the knees*. There may or may not be profuse hemorrhages, either spontaneous or traumatic. In short the patient has an incomplete myxœdema without any marked myxœdematous infiltration of the skin. Given a patient with severe unaccountable dyspnœa, rheumatic pains, and sensation of being cold at all times, a physician should suspect the disease at once. It is evident that the disease is due to a subactivity, or inefficiency of the thyroid gland: (1) because there is an atrophy or fibrosis of the gland; (2) partial thyroidectomy causes it; and (3) it is relieved by the administration of thyroid extract.

In four cases described by Murray, the ages of the patients ranged from forty-four to sixty years. In Moffitt's cases, six in all, the ages at which the disease developed were from thirty-five to sixty-five years. The disease thus is one of middle life. There is sometimes a history of other thyroid disease in the family.

In general the incomplete form bears resemblance to complete myxœdema, but the symptoms are not so profound, nor are there the disfiguring spade-like hands, nor the gross, solid œdema so characteristic of the true form.

In examining a patient with hypothyroidœa the physician is often struck with the disproportion between the cardiac and the pulmonary lesions, or lack of them, and the dyspnœa which is bitterly complained of, and which is often manifest.

In most cases the heart is dilated to some degree; there is, too, a diminution in strength of the first sound. Sometimes there are faint or even marked murmurs depending upon the dilatation.

The cardiac area to the left of the sternum is generally enlarged, the impulse weak, and the pulse may be slow and feeble, or it may be rapid and weak In the case herewith reported the pulse is never below 72 and is sometimes from 110 to 120. One of Murray's patients had a pulse of 80 to 104. Moffitt speaks of a rapid pulse in his cases. Pseudo-angina may be present and with the other neuralgias there may be anginoid pains under the left breast.

The nervous symptoms with the general asthenia are marked and distressing, the giving way of the knees is complained of very much, the patient is tired and disinclined to work. An occipital neuralgia is as often present as frontal headache. Backache, from which the patient suffers greatly, renders her nights sleepless and miserable. Indeed, the patient frequently describes the pain as being so severe that she feels as if her back was being wrenched asunder. The muscles are stiff and the joints painful early in the morning, but generally these symptoms disappear during the day. As in myxœdema the patient feels cold, and is cold, though to the examiner the skin may feel hot and dry. The mental symptoms are sometimes marked. Hallucinations of sight and hearing are common, sound of running water may apparently be heard, visions of cats, dogs, mice, and rats, or of people passing, often occur, even in bright rooms. Murray describes a case in which a marked hallucination was present; it was that of a man knocking a woman down stairs.

The facial expression is one of "sorrowful fatigue." The eyebrows are arched and the forehead wrinkled. Tinnitus aurium also annoys the patient. Murray speaks of his cases as "early thyroid fibrosis" and the thyroid is generally shrunken, hard, and difficult to palpate. The isthmus may have shrivelled up to a thin band. The skin is dry, harsh, and scaly. On removing the stockings a cloud of fine scales is often cast from them. The hair is brittle, dry, and falls out from the scalp, eyebrows, and axillæ. The hair, too, is frequently prematurely gray and white. Subconjunctival fat is yellow. Murray says that bright yellow fat is characteristic of myxœdematous cases and that thyroidectomized monkeys have vivid yellow fat. Above the clavicles are curious pads of fat which are diagnostic, and are almost always present, as in complete myxœdema. There, too, may be rolls or pads of fat above the dorsal vertebræ and over the manubrium. There also may be isolated patches of solid œdema on the abdomen.

The digestive organs are affected in several ways. In common with many asthenic diseases, hypochlorhydria is often present. In a case here described, hyperchlorhydria was a prominent symptom. The appetite is poor, there is always an obstinate constipation and frequently fecal impaction. The liver is enlarged. Transitory glycosuria sometimes occurs. The kidneys are generally sound, but the urine is scanty and high colored. Casts and albumin may appear; hematuria occurs but rarely. The respiratory apparatus is generally unaffected. The lungs may become emphysematous in elderly women. There is often during menstruation a marked huskiness of the voice which is most characteristic. Cramps or cricks of the larynx, probably neuralgic, annoy the patient, and the mucous membrane of the throat and nose becomes thickened.

Spontaneous hemorrhages may occur from any mucous membrane;

menstruation may be scanty or excessive; alarming hemorrhages sometimes occur and if the patient is pregnant, postpartum hemorrhages must be guarded against. Anemia is common in all cases of incomplete myxœdema, just as it is present in every complete form. There is always a diminution of the red cells and the hemoglobin. In both the complete and incomplete cases herein reported, changes in the morphology of the reds were striking; normoblasts, stippled cells, microcytes, and macrocytes were found. It is hard to correlate the many diverse and bewildering symptoms that occur in this protean disease, except through the fact that the *nutrition* of the nervous system, the muscles, and skin is seriously impaired. All of the symptoms depend on insufficient secretion of the thyroid gland, or of certain elements of the secretion. And it may be found that in this incomplete form one or two ingredients of the juice are lacking. The same elements that are of cardinal importance to the nutrition of the skin may not be quite so relatively deficient as those elements that have to do with the nutrition of the brain or peripheral nerves.

When we consider the numerous and highly different elements that constitute the blood plasma, the functions of which are to neutralize poisons, prevent infection, dissolve cells, and aid in the coagulation of the blood, etc., it is not hard to conceive that the growth and nutrition of the body, or that of the skin, or of the brain, depend upon just as diverse elements in the thyroid secretion as complements, hemolysins, and fibrinogens which are found in the blood plasma. If there is singleness of function of the thyroid secretion, it may be that it acts in the same way that Ehrlich's amboceptors act in serving to unite nutritious matters of the blood to cells.

Mrs. K. has the following history of invalidism for sixteen years: She is fifty-five years of age and comes of a healthy family. Her mother is still living, but her father died of sclerosis of the spinal cord. At the age of thirteen she began to menstruate, and continued to do so until she was fifty-two years of age. She has given birth to four large healthy children, and has had the usual diseases of children, pneumonia, and one very bad postpartum hemorrhage. She consulted me for her general condition, which was that of marked asthenia, with a remarkable array of symptoms apparently totally unrelated. She is not corpulent, weighing 145 pounds; is of medium height and build; is active and quick in her movements, and has no disfiguring myxœdema, in fact she is a handsome matron, and except for the anemia, is well nourished. For years she has been afflicted with an occipital neuralgia, which has been at times intractable and violent; she has also suffered from dyspnœa, joint pains, marked myalgias, giving way of the knees, and profuse uterine hemorrhages. A careful physical examination showed a weak musculature, and a harsh dry skin that scaled very easily. Her stockings frequently contained quantities of fine, powdery scales. Above the clavicles oval pads were found, and over the abdomen there was a layer of myxœdematous infiltration. The upper eyelids were swollen and puffy. At times her feet have been œdematous. Her heart dulness was somewhat enlarged to the left, but no murmurs were found. The apex beat was weak and diffuse. The heart was evidently dilated at times.

On account of the dyspnœa, hemorrhages, and weakness of the knees, she had been compelled to remain in bed for many months out of the year. The hemorrhages lasted for three or four weeks, and were difficult to control. An examination by an expert gynecologist revealed a small polyp, the removal of which did not modify the hemorrhages. The uterus otherwise was normal. Her hair turned gray when she was thirty-five years of age, and was brittle and dry, but did not come out easily. Her pharynx and uvula were slightly swollen, and her tongue was indented. At times she has suffered from cramp of the tongue and larynx, and her voice became husky during menstruation. Her pulse was weak and rapid at times, but under thyroid extract its rate diminished. Her vessels were pliable and not atheromatous. She had had a glycosuria of short duration. Her kidneys have always been healthy, although casts were found occasionally. The specific gravity of her urine was 1030, and the amount 40 ounces in twenty-four hours.

It is said that in 20 per cent. of cases of myxœdema, casts and albumin appear in the urine. They often confuse the examiner and he is led to think that the case is nephritis. She has always had a hyperacid stomach, with some tendency to flatulency, and has been constipated, and suffered from impaction at times. Her nervous symptoms were most characteristic; she had, besides the joint pains, backache, and occipital neuralgia, hyperesthesia of the skin of the neck and supraclavicular pads, twitching eyelids, and a slight tremor of the hands, and at night would sometimes tremble for a long time without cause. An examination of her reflexes showed them to be normal. At one time it was thought she had angina pectoris, so intense was the pain in her chest under the left breast. Her joints were so painful that she could not move them, and when she arose in the morning she could hardly step on the floor on account of the pain in her soles. Her rather defective memory was the only sign of any mental involvement. She has never been mentally depressed. At times tinnitus has annoyed her. She sometimes thought she saw people in her room when it was empty. Her nights have been wretched on account of backache, tremblings in her limbs, and achings of the joints, especially of the spine. Her blood showed a moderate anemia: Hemoglobin, 76 per cent.; red cells, 3,268,000; blood index, 1.06; whites not counted. Differential count: Polymorphonuclears, 39 per cent.; lymphocytes, 59.2 per cent.; eosinophiles and myelocytes, 1.8 per cent. Normoblasts, poikilocytes, macrocytes, and microcytes were seen. Numerous red cells exhibiting basophilic degeneration also were found.

Under thyroid extract many of the symptoms were relieved. She cannot tolerate much of any drug. More than 8 grains a day of the extract causes thyroidismus, which is indicated by fever and increased tremor. Under thyroid extract treatment she lost ten pounds in six weeks, but she has taken it at intervals during a year with much benefit. Her dyspnœa is less; her headaches fewer and farther apart, the tremor is gone, and her condition is generally improved. Cold East winds are trying to her still, causing joint pains and occipital neuralgia occasionally. Arsenic, iron, and strychnine have benefited her, as has general massage, which she has taken for years. The constipation was relieved by sodium glycocholate.

COMPLETE MYXŒDEMA. In contrast to the foregoing case the following one of *complete myxœdema* presents not very many differences in the subjective symptomatology, and is a typical case, especially as to the solid œdema.

Mrs. S., a large Englishwoman, aged forty-five years, mother of thirteen children, and of excellent family history, consulted me repeatedly for dyspnœa, and great weakness in the knees. I have attended her for ten years, and delivered her of her last two children. She was always healthy until the birth of her last child five years ago (when forty years of age) when she began to complain of shortness of breath and giving way of her knees. Associated with these two symptoms was a great sensitiveness to cold, even in hot summer weather. After treating her unavailingly for her dyspnœa and weakness, I discovered that she was becoming larger every month. Careful examination showed that she had become myxœmatous. Her hands were very clumsy, her legs and arms and trunk œdematous, but they did not pit on pressure. I could not palpate her thyroid. Her lungs were normal, except for a slight emphysema. Her cardiac dulness had greatly increased and a faint mitral systolic murmur appeared, her pulse was weak and flabby, her dyspnœa was so great that she could hardly walk. Her weight increased from 180 to 208 pounds. Supraclavicular swellings also developed and transitory swellings in the neck appeared over the angle of the jaw. The mucous membranes of the throat and nose became swollen. Her voice was husky, and she had scanning speech. The tongue was swollen and flabby. She complained of constant headache, and was dull and listless. She also had tinnitus aurium. Her expression was one of stupid listlessness, her forehead was wrinkled, and she looked weary and oppressed. Joint pains affected her greatly, as did a constant backache. She could not sleep and she suffered much from cold, and upon putting her hands in cold water they would swell and turn blue and were very painful Her upper incisor teeth were stained black. This is very characteristic of myxœdema.

The urine was normal in every respect, except that it was highly colored, and of high specific gravity. She was anemic: a careful blood count on two occasions showed that she had red cells, 3,344,000; hemoglobin, 75 per cent.; white cells, 13,200 and 6900 on two occasions. A differential count showed: Polymorphonuclears, 46 per cent.; mononuclears, 52 per cent.; myelocytes and eosinophiles, 2 per cent. Stippled red cells were seen in small numbers, as were nucleated red cells. She never had any hallucinations, or severe hemorrhages; nor had she much neuralgia, suffering but twice from trifacial neuralgia.

In September, 1906, she developed a severe bronchitis, and suffered much from dyspnœa, which at that time was due to her dilated weak heart. Dr. M. Howard Fussell saw her in consultation at that time and confirmed the diagnosis of myxœdema. Thyroid extract in daily doses of 15 to 30 grains was administered for a year with very much benefit. Her dyspnœa and nervous symptoms were greatly ameliorated and she was able to move about with great comfort, but was still very myxomatous. Strophanthus and strychnine helped her a great deal. After eighteen months' treatment she became a ruddy, buxom matron again and was bright and intelligent.

The interesting features of this case are the presence of a tremor, absence of hemorrhages, the effect of the cold water on her arms, and great heart weakness.

In both cases the finding of nucleated and degenerated red cells in the blood is of interest, especially when the hemoglobin percentage was not very low. Both cases were in women who had borne numerous children, and both developed the disease when between thirty-five and forty-five years of age. Myxœdema seems to be one of the penalties of childbearing. The weak knees and heart were very prominent symptoms in both cases as was the tremor.

A METHOD OF OBTAINING CULTURES FROM HUMAN BLOOD.

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In making cultures from the living human blood, Lenhartz and Schottmüller advised the mixing of freshly drawn blood with liquified fluid agar (at 45° C.) and the incubation of the culture at 37° C. The method has been employed by them to a large extent and numerous positive blood cultures have been obtained—in sepsis, typhoid fever, pneumonia, and other diseases associated with bacterial infection of the blood. The blood is usually drawn from a vein by means of a Luer's blood syringe, which can easily be sterilized and usually fills by the mere action of the blood pressure. Instead of using the syringe, another simple and less expensive technique may be resorted to which can easily be improvised and gives quite satisfactory results.

A common test tube, such as is used for bacteriological work, is drawn out and bent in the flame of the Bunsen burner until it assumes the shape shown in Fig. 1. It may be drawn out to a sharp point which is sealed in the flame, the open end being closed by means of a plug of absorbent cotton. The tube may contain about 20 c.c. It is sterilized, and immediately before using the sharp point is broken with a file so as to obtain an oval opening wide enough to allow the blood to enter without difficulty. The blood is drawn by means of a wide, sharp-pointed infusion needle as is used for hypodermoclysis, the needle being pushed through the previously well cleansed and disinfected skin into the median cephalic or basilic vein. The pressure within the vein is artificially increased by turning a towel round the arm and thus compressing the brachial vein. The blood readily enters the infusion needle, which should have been previously attached to the test tube by means of a short piece of well boiled and sterilized rubber tubing (Fig. 2) and ascends slowly within about one-half to one minute in the interior of the sterile tube. About 10 to 15 c.c. of blood may be drawn in this way. When the blood level within the tube is about one-half inch below the cotton plug the towel compressing the brachial vein should be released and the needle withdrawn. The thumb of the right hand pressing against the open mouth of the tube above the sterile cotton filter retains the blood by air pressure. The rubber attachment should be removed and the blood immediately distributed into five or six tubes containing liquified plain agar or glycerin-agar of a temperature of 44° C., and thoroughly mixed with it by rapidly twirling the tubes between the hands. The mixture then should be put into Petri dishes before the agar solidifies, and the blood culture transferred to the thermostat as quickly as possible.

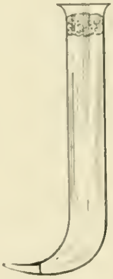

Fig. 1.—Test tube drawn out and bent for blood culture work.

In this way I examined the blood of six patients in the King's County Hospital. In 3 cases the blood-agar plates remained sterile, in the rest I obtained a positive bacteriological finding. The first of these cases was a grave sepsis caused by a neglected phlegmon of the right leg. The patient was received in a hopeless state, running a high continuous fever, markedly jaundiced, and died after a few days. The blood culture which was taken two days before death revealed in three agar-plates (about 7 c.c. of blood) the presence of streptococci, which could be seen as pure colonies, in number about 50, after thirty-six hours' incubation. The colonies appeared as small blackish spots within the blood-agar; they showed whet-stone form, and were characterized by a colorless halo about 2 mm. in diameter, within the limits of which the hemoglobin of the red blood cells had disappeared.

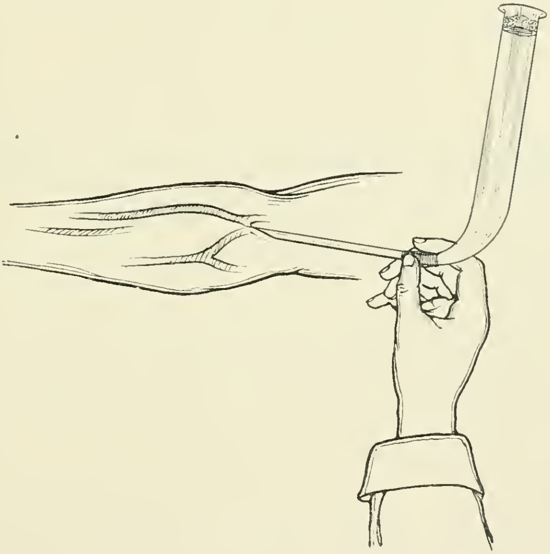

FIG. 2.—Withdrawing the blood for cultural purposes.

The second case was one of puerperal sepsis. The patient was received in the hospital running a continuous high temperature, about 104° F. When the blood culture was made the skin of the breast, legs, and arms showed an extensive erythematous rash, in appearance very much reminding one of scarlatina. The rash disappeared after about twenty hours. The culture showed, after twenty-four hours incubation, numerous colonies of streptococci, and colonies of another kind, appearing as blackish spots within the agar, which, rapidly extending to the surface, spread as yellowish well-circumscribed, moist, and shining round colonies (Staphylococcus pyogenes aureus). Twenty-four hours after the culture had been taken, the patient died.

The third case was one of typhoid fever, toward the end of the second week. The temperature was between 103 and 104° F. The blood culture showed, in 10 c.c. of blood, 102 pure colonies of typhoid bacilli. The typhoid colonies appeared as blackish spots and points, visible after twenty-four hours incubation. Within the next twenty-four hours part of them reached the surface where they spread as a well-circumscribed shining growth, showing a peculiar grayish (mouse-gray) color.

The pneumococcus has been found by Lenhartz in grave cases of pneumonia in 20 per cent. of the cases. As to the appearance of its growth it may only be mentioned here that the macroscopic appearance of the colonies in blood-agar plates closely resembles that of the streptococcus varieties, but the halo of decoloration surrounding the single colonies which appear as black wheat-stone shaped spots of about 0.5 mm. in length is not clear as in the case of the streptococcus, but shows a distinct greenish hue.

OBSERVATIONS UPON CERTAIN BLOOD-PRESSURE-LOWERING REFLEXES THAT ARISE FROM IRRITATION OF THE INFLAMED PLEURA.

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Thoracentesis and all operative procedures inside the thoracic cavity where inflammation exists may be accompanied or followed by disturbances of circulation. These occur even when such complications as pulmonary œdema, accidental hemorrhage, and pneumothorax can be definitely excluded. As a rule these disturbances are trivial and consist of lowering of arterial tension accompanied by faintness. Occasionally, however, the symptoms are serious. Without warning the patient becomes dyspnœic and cyanotic, and the pulse is thready, with a tension below the safety point. Unconsciousness and death may supervene in a few minutes or in a few hours.

The cause of death in these cases is usually not discovered by the pathologist. Rarely emboli or thrombi are found in the heart or pulmonary arteries and even more rarely in the cerebral arteries

(Rosenbach). After excluding such exceptional conditions there still remains a considerable number of cases of sudden death in which *no anatomical lesion can be found postmortem to explain the collapse.*

Rosenbach[1] considers that the chief factors in producing death in these obscure cases are anemia of the heart or brain, sudden heart-failure, or a reflex inhibition of the respiratory and cardiac centres. Russell[2] believes that the pulmonary fibers of the vagus nerve, made sensitive by inflammation, carry impulses to the medulla. The impulses originate, he thinks, in the alveoli and not in the pleura.

In a series of recent observations by one of us[3] upon the blood pressure in aspirating pleural effusions, it was concluded that the *rapidity of withdrawal*, the removal of a *large amount* of fluid, the presence of a *long-standing effusion* and of *senile changes* in the heart and bloodvessels, were the chief factors in bringing about a marked fall of arterial blood pressure with unfavorable symptoms. All these, however, are contributing causes only and fail to explain the mechanism of the phenomena.

The following experiments were made with the purpose of finding out if possible the nature of this mechanism:

GROUP I.—*Aspiration of Oil from the Normal Pleural Cavity.* All the dogs under this group were injected in one pleural cavity with sterile cotton-seed oil, which, unlike watery solutions, is not absorbed by the pleural membrane. The fluid was allowed to flow in until the first signs of dyspnœa developed. Readings of the blood pressure were taken before, during, and after aspiration.

EXPERIMENT 1. 700 c.c. cotton-seed oil injected into left pleural cavity of large dog. 500 c.c. withdrawn in the period of five minutes:

Initial blood pressure	130 mm. Hg.
After injection of 700 c.c. of oil	140 "
Immediately after aspiration of 500 c.c.	115 "
Final blood pressure ten minutes after aspiration . . .	120 "

EXPERIMENT 2. Medium-sized dog:

Initial blood pressure	132 mm. Hg.
After injection of 500 c.c. of oil in left pleural cavity . . .	138 "
After aspiration of 400 c.c.	134 "
Final blood pressure eight minutes later	130 "

EXPERIMENT 3. Large dog:

Initial blood pressure	128 mm. Hg.
After injection of 750 c.c. of oil in right pleural cavity . .	148 "
After aspiration of 550 c.c.	140 "
Final blood pressure five minutes later	125 "

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[1] Nothnagel's Encyclopedia of Medicine, 1903.

[2] St. Thomas' Hospital Reports, 1899, vol. xxviii.

[3] Capps, Jour. Amer. Med. Assoc., January 5, 1907.

EXPERIMENT 4. Rather small dog:

Two days after injection of 450 c.c. sterile oil in right pleural cavity	100 mm. Hg.
After rapid aspiration of 350 c.c.	120 "
Final blood pressure	100 "

No pleurisy found postmortem.

EXPERIMENT 5. Medium-sized dog:

Initial blood pressure	70 mm. Hg.
After injection of 600 c.c. of oil in right pleural cavity	80 "
After aspiration of 450 c.c	70 "
Final blood pressure ten minutes later	70 "

EXPERIMENT 6. Dog of large size:

Initial blood pressure	100 mm. Hg.
After injection of 600 c.c. of oil in left pleural cavity	110 "
After aspiration of 350 c.c.	95 "
Final blood pressure ten minutes later	97 "

Discussion. The average rise in blood pressure in these six cases produced by injecting the oil was about 12 mm. Hg. The average fall in pressure as a result of aspiration was 11 mm. Hg. The rise in pressure accompanying the introduction of fluid may be attributed to the diminution of lung space and to pressure exerted by the weight of the fluid on the lungs, the heart, and the neighboring vessels. The fall in pressure following aspiration may be referred to the restoration of physiological conditions. There was no evidence of any reflex disturbance of circulation during aspiration, such as transitory falls in pressure or changes in the pulse rate.

GROUP II.—*Aspiration of Exudates in Artificial Pleurisy.* The pleurisy was created by injecting from 10 to 300 c.c. of cotton-seed oil contaminated with bacteria in one pleural cavity of a dog. At the end of twenty-four to forty-eight hours a bilateral pleurisy had developed, with a thick pleural exudate and a large amount of free fluid on the injected side; on the opposite side, a thinner pleural exudate and very little free fluid. The fluid was aspirated with a water pump as rapidly as possible.

EXPERIMENT 7. Bilateral pleurisy in a dog which had been injected forty-eight hours before with 10 c.c. of dirty oil in the right chest:

Blood pressure before aspiration	130 mm. Hg.
After aspiration of 150 c.c. of oil and exudate	110 "
Ten minutes later	115 "

EXPERIMENT 8. Dog with marked right pleurisy twenty-four hours after injection of 200 c.c. of oil in right side; left pleura congested:

Blood pressure before aspiration	90 mm. Hg.
After aspiration of 150 c.c.	70 "
Six minutes later	74 "

EXPERIMENT 9. Dog injected forty-eight hours previously in right pleural cavity with 300 c.c. of dirty oil. Right pleurisy moderate, left slight:

Initial blood presssure	110 mm. Hg.
After aspiration of 200 c.c.	110 "
Five minutes later	110 "

Experiment 10. Bilateral pleurisy in dog injected in left side forty-eight hours before with 225 c.c. of oil:

Initial blood pressure	130 mm. Hg.
After aspiration of 150 c.c.	130 "
Six minutes later	130 "

Experiment 11. Bilateral pleurisy forty-eight hours after introduction of 200 c.c. of oil in right pleural cavity:

Initial blood pressure	110 mm. Hg.
After aspiration of 150 c.c.	100 "
Eight minutes later	105 "

Experiment 12. Bilateral pleurisy forty-eight hours after injection of 200 c.c. of oil:

Initial blood pressure	120 mm. Hg.
After aspiration of 100 c.c.	100 "
Five minutes later	105 "

Experiment 13. Bilateral pleurisy forty-eight hours after introduction of 200 c.c. of oil into right cavity:

Initial blood pressure	140 mm. Hg.
After rapid aspiration of 100 c.c.	30 "
Three minutes later	Death.

During the process of aspiration in this experiment the respiration slowed and finally ceased altogether, although the ether was withdrawn.

Discussion. It should be noted that the average amount of exudate withdrawn in this group was about 150 c.c., while the average quantity in Group I was about 500 c.c. We should expect less of a fall in pressure, therefore, in Group II from purely pressure effects. As a matter of fact, the results were about the same in the two groups, which would indicate that aspiration of a small quantity of fluid from an inflamed pleural cavity may produce as much lowering of tension as a much larger quantity of fluid from a normal cavity. Transitory changes in pressure were common during these experiments, and especially in the last one it was observed that the movement of the trocar on the pleura sometimes caused a marked fall in blood pressure, even when the amount of fluid aspirated was small. We were thereby led to investigate the effect produced on blood pressure by irritation of the pleura, healthy and inflamed, independently of aspiration.

Group III. *Irritation of Healthy Pleura.* The procedure in these experiments was as follows:

The dog was etherized and laid on the operating table. The trachea was opened and a cannula inserted which connected with a flask of ether. By means of a T-glass cannula in this connecting tube a smaller tube was attached that carried the air waves to a recording

It may be noted that the winds around the Great Lakes are especially high during the winter months and become somewhat lower during the months of June, July, August, and September. And not only are the winds of higher velocity in miles per hour, but examination of Fig. 2 will demonstrate that the total wind movement in miles per month is much greater in the cities of the Great Lake region than in the inland cities.

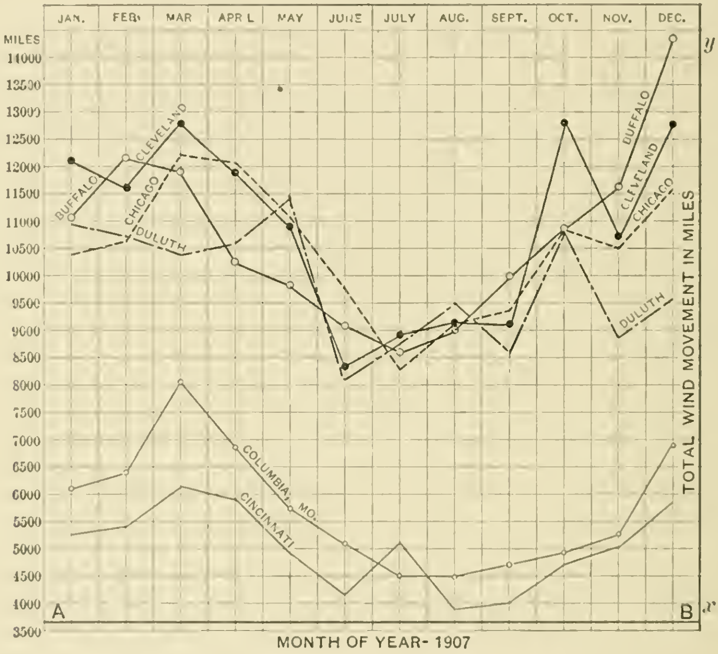

FIG. 2.—Total wind movement in miles per month.

To summarize, the winter climate around the Great Lakes, though not showing extreme changes in temperature in twenty-four hours, is very variable, especially in conjunction with the winds of high velocity and of high wind movement in miles per month.

CASE HISTORIES.

CASE I.—Female, aged twenty-three years; occupation housework. Aside from the present trouble the patient has always been well and strong. About two weeks before admission she first began to notice an itching, burning eruption which broke out on the back of one hand. Since then it has bothered her a great deal, and she has noticed a moist exudate at times. At the time of writing there is an area about 2 x 2 cm. on the back of the left hand, and a smaller one on the right wrist. Both areas are circinate, well defined, with slightly elevated edges, and a tendency to heal in their centres. As yet there is verly little crust formation, but one sees some cracking of the surfaces and a little serous exudate. The areas are of a dark red color and show no induration. Patient says none in the family are troubled with a like affection, and she has never had it before.

Case II.—George S., aged twenty-one years; switchman. The family and personal histories are negative. Patient had an infection of the second finger of the right hand three years ago, and ever since there has been a scaling and cracking of the skin over this area. About three months before admission an eruption broke out at the base of the third finger of the left hand and the like finger of the right. The lesions have extended a little, and a month later another lesion appeared on his right forearm. Patient was admitted to Lakeside Hospital for study. The blood showed at that time 3 per cent. of eosinophiles, otherwise nothing out of the ordinary was found. The lesions were similar to those of Case I except that there was crust formation and more induration of the areas. A biopsy was made of several fresh papules found on the affected area of the forearm. The finger involved for three years showed merely a chronic eczema, illustrating the fact that an eczema and a dermatitis hiemalis may be present on the same patient.

Case III.—Carl W., aged thirty years; baker. Patient is a Bohemian, and was never troubled with this disease until last year, when he came to this country and settled in Detroit. The disease was present all winter, disappearing the summer following, only to appear again the next October. Patient came to Cleveland, hoping to be relieved of the trouble, but he has been troubled off and on for the last five months. The eruption itches and burns a great deal, and there is more or less of a moist exudation. The areas bleed quite easily; otherwise he feels perfectly well. Both posterior surfaces of the hands and one wrist are affected with well-defined, circinate, dark red lesions, showing an elevated edge and a noticeable tendency to heal in their centres. Crust formation is quite marked, and there is some induration of the parts. A biopsy was made of an area about three or four weeks old, and also from a spot of more recent appearance.

Case IV.—G. W. P., aged thirty-five years; bookkeeper. His habits are good, and he has always been well, except for the present trouble. For the last ten years the patient has been bothered every year with an itching, burning eruption, which is limited to the hands, and is present only during the winter. It appears early or late in the fall, according to the severity of the weather, as he has himself noticed, and lasts until spring. At times the lesions will partially heal, but only to reappear, and he has never found anything that gave him much relief. He says he has never changed climate during the winter, so does not know what effect another climate would have on the condition. No others of the family are

200 c.c. of cotton-seed oil contaminated with bacteria. In twenty-four hours the pleural surface was covered with a fibrinous film and a few flakes were often found on the opposite side. After forty-eight hours the inoculated side presented a dense, fibrinous exudate with fluid accumulation, while the opposite pleura was overlaid by a thin, fibrinous exudate, accompanied by a variable amount of free fluid.

Since the chemically produced pleuritis develops more quickly, animals so treated were employed from six to twenty-four hours after injection. The animals inoculated with the more slowly developing bacterial injection were subjected to experiment twenty-four to seventy-two hours after the injection.

It seemed desirable to test the pleura under various degrees and varieties of inflammation as well as to compare in the same animal the effect of irritation of the highly inflamed pleura of one side with the slightly inflamed pleura of the other side.

(*A*) *Chemical Pleuritis from Injection of Turpentine.*

EXPERIMENT 23. A dog had been injected on the left side twenty-four hours previously with 3 c.c. of turpentine mixed with 9 c.c. of sterile oil. Animal appeared languid. Ether anesthesia. A mercury manometer was connected with the carotid artery and the respiratory tambour with the breathing cannula, which was inserted in the trachea. After the ether was evenly regulated, a trocar was forced into the right chest in the fifth interspace in the midaxillary line, and the sharp point retracted. The visceral pleura was then scratched with the blunt end of the trocar, with no change in blood pressure. The sharp point also caused no change in pressure when scraped along the visceral or parietal pleura. Three drops of formalin blown through the trocar caused no change in blood pressure, although respirations were slightly slowed. No effect was observed from the trocar irritation of the left visceral and parietal pleura, but the introduction of 2 drops of formalin caused a sudden slowing of the heart with long strokes of the pen (cardio-inhibitory or "vagal" type), with simultaneous slowing of respiration. The effect lasted for a little over a minute (Fig. 2). The chest was then opened by bone-cutting forceps in the median line, and the thoracic viscera exposed. Respirations were maintained by an air pump. This latter procedure lowered the blood pressure from 100 mm. to 70 mm., at which level the tracing remained steady. Mechanical irritation of left and right parietal and visceral pleuræ with blunt and sharp point had no effect on blood pressure. Scratching the pericardium "in situ" set up long drops of "vagal" type, but when the sac was held away from the heart, the irritation had no effect. Laceration of the lung tissue by squeezing in forceps was negative. Electrical irritation (faradic) of left and right visceral and parietal pleuræ was negative. Stimulation of pericardium "in situ" gave "vagal" type of curves identical with those following direct stimulation of the heart muscle, but stimulation of the free pericardium was negative. Chemical irritation of both visceral and parietal pleuræ had no influence on the pressure curve. Thermal irritation by hot iron and ice of left and right parietal and visceral pleuræ did not alter blood pressure. Briefly stated, the only reflex obtained was through irritation of the left pleura by formalin, and this reflex was lost after opening the chest. Postmortem a fairly marked exudate in left side, with fibrinous pericarditis, was found. Right pleura was normal.

Experiment 24. A dog had been injected twenty-four hours previously in left side with turpentine 2 c.c. and oil 10 c.c. Trocar irritation of both left and right visceral pleuræ, negative. Formalin 2 drops on right visceral pleura caused slowing of respiration and slight fall in blood pressure. The same amount of formalin on left visceral pleura caused slowing and temporary stopping of respiration, with decided fall in blood pressure (20 mm.). Recovery in about one minute. Postmortem: Thin layer of lymph and small amount of purulent exudate on left side, right pleura congested, no exudate.

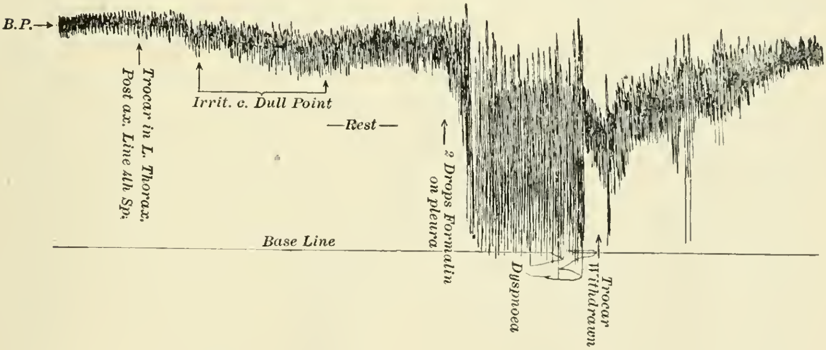

Fig. 2.—*Cardio-inhibitory Reflex.* The upper tracing represents the blood pressure; the base line is below. The inflamed pleura was irritated first with the trocar, then with two drops of formalin. Note the sudden appearance of long and slower strokes after the formalin. Recovery was spontaneous.

Experiment 25. Left side of chest of dog injected eight hours previously with turpentine 4 c.c. and oil 8 c.c. Blood pressure, 160 mm. Hg. Right visceral pleura repeatedly irritated with sharp point of trocar caused slight slowing of respiration, frequent vagal curves, with a slowing of heart and steady fall in blood pressure to 120 mm. Hg. in the course of two minutes. Rapid recovery to 130 mm. when irritation ceased. Trocar irritation of left visceral pleura slowed respiration slightly, but made no appreciable change in blood pressure. Trocar irritation of right visceral pleura was repeated with fall in blood pressure from 165 to 140 mm. Then both vagi were cut in the neck. The blood pressure fell to 120 mm., then remained steady while respirations were slower. Further irritations of right pleura caused steady fall in blood pressure to 110 mm. with "vagal" strokes. Formalin 4 drops on right visceral pleura negative. Formalin 4 drops on left visceral pleura caused immediately respiratory paralysis and a rapid fall in blood pressure, with death. The cardio-inhibitory curves were set up especially by irritation near root of lungs. Section of vagus nerves failed to destroy entirely the cardio-inhibitory curves or the vasomotor fall. Postmortem: Left pleurisy with thin layer of fibrin and small amount of fluid. Right pleura congested. The abdominal viscera were engorged with blood.

Experiment 26. Dog was injected six hours before in right chest with turpentine 2 c.c. and oil 6 c.c. Blood pressure, 90 mm. Trocar irritation of left visceral pleura negative. Trocar irritation of right visceral pleura was followed by slight lowering of blood pressure from 90 mm. to 80 mm. and respirations unchanged. Postmortem: Fine fibrinous threads over right pleura. Left pleura normal.

Experiment 27. Six hours before experiment a small dog was injected in right chest with turpentine 4 c.c. and oil 4 c.c. Blood pressure 100 mm. Trocar irritation of right visceral pleura was negative. Trocar irritation around root of right lung caused the cardio-inhibitory curves. Trocar irritation of left visceral pleura was negative. Formalin 5 drops on left visceral pleura was negative. Formalin 5 drops on right visceral pleura followed by slower and more shallow respirations and by a fall in blood pressure to 76 mm., with long curves. Recovery in about two minutes. Postmortem: Thin pleuritic exudate on right side. Left pleura normal.

Experiment 28. Dog injected twenty-four hours before in right pleural cavity with turpentine 1 c.c. and oil 6 c.c. Blood pressure, 118 mm. Hg. Trocar irritation of right visceral pleura caused a slight fall in blood pressure. Trocar irritation of left visceral pleura was negative. Formalin 5 drops on right visceral pleura was followed by a gradual rise in pressure from 120 mm. to 134 mm. Formalin 5 drops on left visceral pleura was negative. Postmortem: Right pleuritis with thick fibrinous exudate and free fluid. Left pleura normal.

(B) *Bacterial Pleuritis of Twenty-four Hours or Less After Injection of Contaminated Oil.*

Experiment 29. Twenty-four hours previously dog received injection in the left chest of 5 c.c. of contaminated oil. Blood pressure, 122 mm. Trocar irritation with sharp and dull point of right visceral pleura was negative. Trocar irritation with dull point of left visceral pleura was negative. Trocar irritation with sharp point of left visceral pleura caused a rapid fall in blood pressure from 110 mm. to 64 mm., with shallow respirations. Recovery in

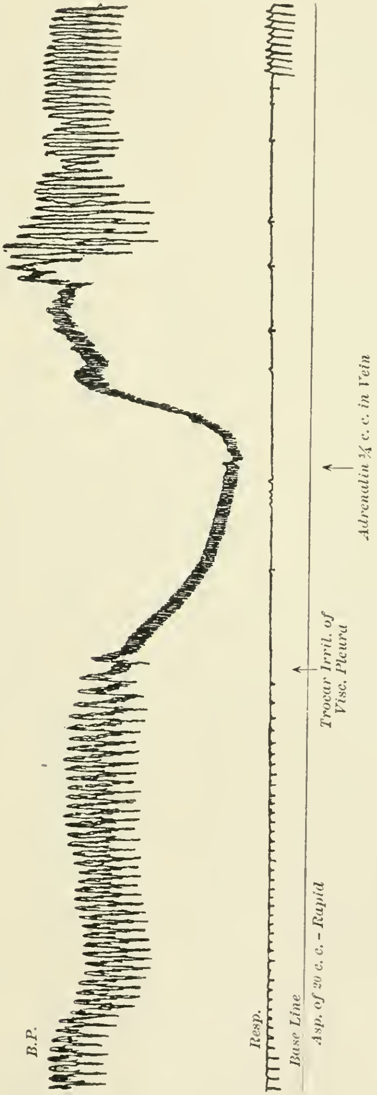

FIG. 3.—*Vasomotor reflex* resulting from scratching the inflamed visceral pleura with a sharp point of the trocar. Respirations ceased simultaneously with the fall in blood pressure. Adrenalin intravenously caused rapid recovery of pressure with gradual return of respirations. Note also the moderate fall in pressure from the rapid withdrawal of fluid.

two minutes to 80 mm. An injection of 0.5 c.c. of adrenalin in the femoral vein was followed at once by a rise in blood pressure to 200 mm. and the animal recovered (Fig. 3.) Formalin drops 5 on right visceral pleura was negative. Formalin drops 5 on left pleura caused a slight fall in pressure. Postmortem: Right pleura normal. Left visceral pleura showed a fringe of dense fibrin at edge of lobes, smaller flakes over surface and 100 c.c. of fluid. Evidence of trauma from trocar on middle and upper lobes about an inch from right bronchus. No puncture of lung could be detected.

Experiment 30. Twenty-four hours previously 5 c.c. of contaminated oil had been injected into the left pleural cavity of a small dog. Trocar irritation with dull and sharp point of right visceral pleura, negative. Trocar irritation with dull and sharp point of left visceral pleura, negative. Formalin 3 drops on right visceral pleura, negative. Formalin 3 drops on left visceral pleura caused a slight fall in blood pressure. With the chest laid open and artificial respiration the same tests were repeated with negative results. Formalin injected directly in the lung tissue on both sides, and compression of lung tissue in the forceps, also were without effect on blood pressure. Post-mortem: Left pleurisy with moderate amount of exudate. Right pleura normal.

Experiment 31. Into the right chest of a dog 10 c.c. of contaminated oil had been introduced twenty-four hours before experiment. With closed chest the visceral pleura was irritated on both sides with trocar and formalin. Except for a slight fall in blood pressure after applying formalin to the left pleura, the result was nil. Postmortem: Fairly advanced pleurisy on right side. Left pleura normal.

Experiment 32. To a young dog 8 c.c. of dirty oil was injected in the right thorax twenty-four hours previous to experiment. Scratching the pleural surfaces caused no depression of blood pressure. Application of 5 drops of formalin to the right pleura was followed by a slight fall in blood pressure and slowing of respiration. Both vagi were then cut in the neck. After a period during which the pressure became steady again, 0.5 c.c. of formalin was again applied to the right pleura. Immediately respirations were slowed and the blood pressure fell with long curves from 110 mm. to 40 mm. Recovery was spontaneous. The reflex was not pure vagal, but showed a vasomotor fall as well. Postmortem: Right pleura very congested, but no distinct membrane. Free oil and some purulent exudate in cavity. Left pleura normal.

Experiment 33. Dog similarly injected in right pleural cavity twenty-four hours before with 200 c.c. of contaminated oil. Rapid aspiration of 150 c.c. of fluid caused a fall in blood pressure from 90 to 70 mm., with gradual recovery to 80 mm. Irritation of right visceral pleura with the trocar produced little change in pressure. Postmortem: Right pleura dotted with fibrin flakes and over 100 c.c. of free fluid present. Left pleura was congested.

(*C*) *Bacterial Pleuritis Forty-eight Hours or More After Injection of Contaminated Oil.*

Experiment 34. Dog prepared forty-eight hours before by introduction into left chest of 400 c.c. of dirty olive oil. Blood pressure at beginning of experiment was 150 mm. A trocar was inserted in the fifth space, mid-axillary line of the right pleural cavity. Only 2 or 3 c.c. of fluid had escaped when the blood pressure fell rapidly to 56 mm. and the respirations became shallow. Upon withdrawing the trocar the blood pressure quickly went up to 106 mm. when the trocar was reinserted. Again the pressure fell, soon reaching the abscissa line and terminating in death. The trocar could be felt against the visceral pleural membrane, but the amount of friction was not great. Respirations were shallow and slowed, but continued after the pressure reached zero. Postmortem: Left pleural cavity contained about 300 c.c. of oil and purulent exudate. Pleural surface covered with a thick lymph layer. The right cavity held about 50 c.c. of fluid and a thin layer of fibrinous exudate on the lung, which showed two points where the trocar had scratched the pleura. There was slight ecchymosis, but no rupture of the pleura and no evidence of bleeding in the branchioles or pleural cavity. The abdominal viscera were congested to a high degree.

Experiment 35. Forty-eight hours previously 450 c.c. of dirty oil had been injected into the left thorax of medium-sized dog. Thoracentesis in right fifth interspace, anterior axillary line. When the sharp point of the trocar was moved over the visceral pleura a steady fall in blood pressure took place and continued until death, which occurred in four minutes. Respirations were shallow and slowed, but continued until blood pressure was nearly zero. Postmortem: Left pleura overlaid with a thick fibrinous layer and in the cavity was about 300 c.c. of oil and pus. The right side contained about 100 c.c. of oil and the pleura was thinly concealed by a lymph layer. Near the root of the lung the pleura was discolored as well as posteriorly by the trocar scratch, but no perforation was present. The bloodvessels of the stomach, intestines, and liver were abnormally congested. Brain normal.

Experiment. 36. For three days 300 c.c. of dirty oil had been present in the left pleural cavity. Irritation of the outer surface of the left visceral pleura with the trocar was negative, but when the trocar scratched the pleura near the root of the lung long "vagal" strokes were produced. Cutting both vagus nerves in the neck seemed to modify these curves, but did not prevent them altogether. Postmortem: Over 300 c.c. of purulent oil in left cavity, with thick plastic layer on pleura. Right pleura also covered with a thick, soft exudate and about 40 c.c. of free fluid in the cavity. No perforation of lung and no hemorrhage was found.

Experiment 37. Dog had received an injection two days before in left chest of 200 c.c. of dirty oil. A trocar in right pleural cavity in fifth space, anterior axillary line, when moved laterally caused long vertical strokes on the drum, without much influence on the average pressure. On the left side scratching with the trocar was without effect. Postmortem: In the left side of chest was over 200 c.c. of free fluid and a dense pleural membrane. The right lung was covered with a thinner layer of exudate. The heart was displaced to the left. Possibly the trocar touched the pericardium in its movements and thereby set up "vagal" curves.

Experiment 38. The right thorax of a dog had been injected two days before with 300 c.c. of dirty oil. Trocar irritation of right visceral pleura after withdrawal of 200 c.c. of oil caused practically no change in blood pressure. Trocar irritation of left pleura was followed by a slight fall in pressure. Formalin 2 drops on the left pleura caused great cardio-inhibitory curves, with fall in blood pressure which continued for nearly a minute. Respiration was slowed and labored. Recovery. Postmortem: Right pleura and the pericardium were covered with a thick fibrinous membrane. The left pleura was inflamed and overlaid with a thin, translucent membrane of lymph. The lung tissue puckered where formalin had come into contact with its surface.

Experiment 39. Dog was injected in the right pleural cavity with 50 c.c. of dirty oil. Mechanical and chemical irritation of pleura were negative. With the chest opened and under artificial respiration, all forms of irritation, mechanical, chemical, thermal, and electrical, were applied to various parts of the pleura with no influence on blood pressure, except over the roots of the lungs, where mechanical and electrical stimulation produced occasional cardio-inhibitory strokes without altering the general level of blood pressure. Postmortem: Right thorax contained about 150 c.c. of turbid oil and over the pleura was a thin, soft exudate. Left lung retracted from pressure of 100 c.c. of fluid and covered with flakes of fibrin. Pericardium concealed by a thick exudate.

Experiment 40. Dog prepared two days before by introduction of 30 c.c. of contaminated oil into the right pleural cavity. Animal was lively. Stimulation of both pleuræ with the trocar caused only the long strokes that occur physiologically from sweeping along the roots of the lung. Formalin had no effect. Postmortem: Very little inflammation was found in the right side of the thorax and only 50 c.c. of oily fluid. The left pleura appeared normal.

Experiment 41. Dog forty-eight hours before was injected with 10 c.c. of dirty oil in left chest. Trocar irritation of right visceral pleura with dull point caused only a slight lowering of blood pressure from 126 mm. to 120 mm., but upon scratching the surface with the sharp point the pressure fell rapidly until death took place two minutes later. The respirations simultaneously were slowed and when the pressure reached 50 mm. ceased entirely. Cutting of both vagus nerves in the neck had no apparent effect on the blood pressure (Fig. 4). Postmortem: Pleurisy of high degree on left side, with a thick exudate over the lung. Right pleura much congested, with a few shreds of fibrin on the surface. Slight pericarditis. Evidence of trocar scratches on the right pleura about one inch external to the root of the lung. No perforation of the lung was found.

Experiment 42. In a dog 10 c.c. of dirty oil had been introduced into the left pleural cavity forty-eight hours previously. Irritation of pleuræ on both sides with sharp and dull point produced no change in blood pressure. Formalin injections on the pleuræ were likewise negative. Formalin 1 c.c. sprayed in trachea and bronchial tubes caused a gradual deep fall in pressure from 120 to 6 mm., with slower and deeper respirations. Spontaneous recovery. Postmortem: Left pleura covered with thick layer of lymph and in the cavity 100 c.c. of fluid. Right pleura red and streaked with a few fibrin shreds.

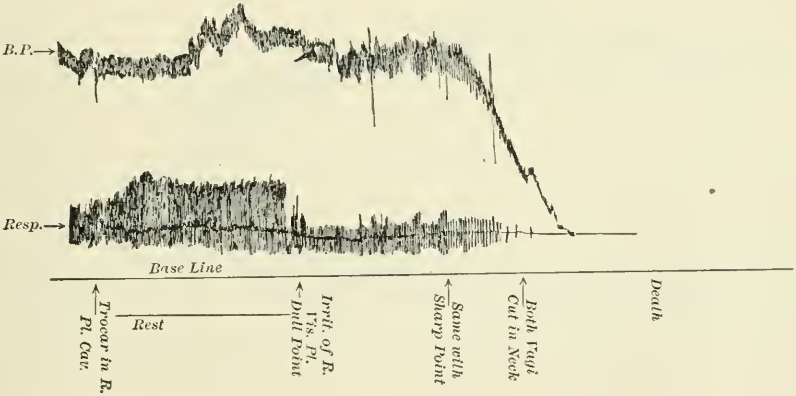

Fig. 4.—*Vasomotor reflex* following irritation of the visceral pleura with a sharp trocar. At the bottom is the base line, the respirations, and at the top the blood pressure tracing. Note rapid fall of pressure and slowing of respirations until death. Section of both vagi in the neck had no effect on blood pressure. Engorgement of abdominal viscera postmortem.

Experiment 43. The dog was injected forty-eight hours before with 8 c.c. of dirty oil in the right pleural cavity. Trocar irritation with dull point of right visceral pleura caused a slight fall in pressure, of the left pleura no change except occasional vagal strokes. Formalin 5 drops on left pleura caused a slight fall in pressure with shallow respirations. The sharp point of the trocar moving over the surface of the left visceral pleura caused slowing of respiration, long "vagal" dips, and a steady fall in pressure from 135 mm. to 80 mm. Then both vagi were cut in the neck without marked effect. By discontinuing the irritation of the pleura the pressure gradually recovered to 110 mm. Further irritation caused shallow breathing and a rapid fall in blood pressure that terminated in death in a minute and a half. Section of the vagi did not prevent the vasomotor reflex. Postmortem: Right pleura covered with thick exudate. Left pleura inflamed and overlaid by a thin film of fibrin. Evidences of trocar scratches and very slight ecchymosis, no perforation. Congestion of abdominal vessels was marked. Brain normal.

EXPERIMENT 44. Dog used forty-eight hours after 10 c.c. of dirty oil was introduced into the right pleural cavity. Trocar irritation of right visceral pleura (dull point) caused a moderate fall in blood pressure, with slowing of breathing. By drawing the blunt point over the region of the sympathetic chain, respiration was slowed, then paralyzed, and the pressure curve showed long vagal sweeps with a rapid fall in tension from 100 mm. to 40 mm. Cutting the left vagus had no effect. When the right vagus was also cut the vagal sweeps disappeared and the pressure fell rapidly until death ensued a minute later; that is, the cardio-inhibitory reflex was abolished by section of both vagi, but the vasomotor fall in pressure continued. (Fig. 5). The cardio-inhibitory reflex was therefore central and the impulse to the centre was carried by the vagus nerves. Postmortem: Trocar left "in situ" was found to be pressing directly on the thoracic sympathetic nerve in the sixth space. Also marks of trocar on pleura of middle lobe. Vessels of stomach, intestines, liver, etc., much congested.

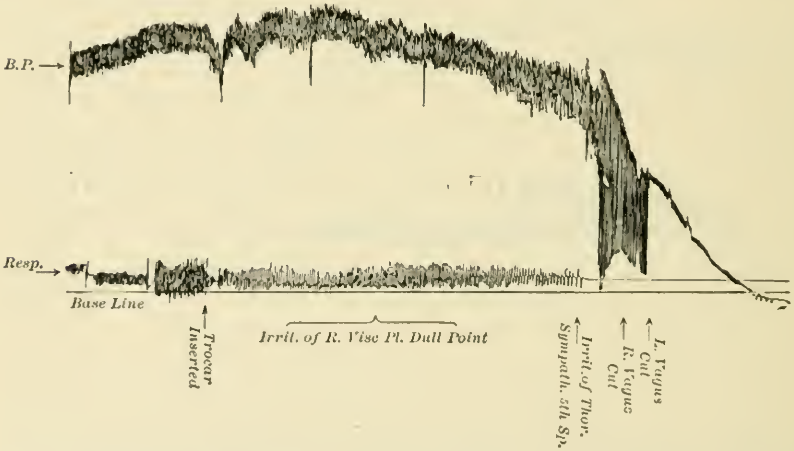

FIG. 5.—*Combined cardio-inhibitory* and *vasomotor reflex* from irritation with a trocar of right thoracic sympathetic nerve in a dog with pleurisy. Upper tracing is blood pressure, lower is respirations, and at bottom the base line. Note gradual fall in pressure from irritating visceral pleura with a dull trocar, then rapid fall with long strokes and cessation of respiration when the nerve trunk is irritated. Section of both vagi in the neck eliminated the cardio-inhibitory strokes, but had no influence on the steady vasomotor fall. Death. Note also the transitory fall in pressure and suspension of respiration when the trocar was forced through the chest wall.

Experiment 45. The dog forty-eight hours previously had been given 10 c.c. of dirty oil in the right chest. Aspiration of the exudate was accompanied by a fall in pressure from 120 to 110 mm. Trocar irritation of the right and left visceral pleuræ was negative. Formalin 1 c.c. on the right pleura was followed by a slight fall; 1 c.c. of formalin in the left membrane caused a slight rise, then rapid fall from 130 mm. to 14 mm. The type of curve was vasomotor. During the descent of the blood pressure both vagi were cut in the neck, but no effect was seen; 0.5 c.c. of a 1 to 1000 solution of adrenalin injected into the femoral vein caused an instant rise from 16 mm. to 210 mm. Final recovery. During the low tension the respirations were slow and shallow. Postmortem: Right pleurisy marked with thick membrane over lung. Left side contains a small amount of fluid and a beginning pleurisy.

Experiment 46. Dog prepared by injecting, forty-eight hours previously, 225 c.c. dirty oil in the left pleural cavity. 150 c.c. of fluid aspirated from left side. Trocar irritations of left visceral pleura were negative. Trocar irritation of right visceral pleura showed a gradual fall from 120 mm. to 110 mm. Formalin 1 c.c. in right pleura caused a fall in blood pressure from 116 mm. to 80 mm., with shallow respirations. Recovery. Postmortem: Double pleurisy, more marked on left side.

Experiment 47. Injection of 200 c.c. of oil in right pleural cavity of dog forty-eight hours previously. 150 c.c. of purulent fluid was removed by aspiration. Atropine 1 mg. was given intravenously, until electrical stimulation of vagus caused no change in the heart action. Formalin 1 c.c. in right visceral pleura caused a vasomotor type of fall in pressure, with slower and shallower respirations. Cutting vagi had no effect on the curve. Death. Postmortem: Right pleurisy, with thick, fibrinous covering. Left pleurisy starting. Engorgement of abdominal viscera.

Experiment 48. Double pleurisy created in right side of dog by injecting forty hours previously 200 c.c. oil. 100 c.c. exudate aspirated from right side. Formalin 5 drops on right visceral pleura within a minute set up long strokes, with slowing of respiration. At first the blood pressure rose, then fell. Atropine 1 mg. in femoral vein caused an immediate cessation of the "vagal" strokes. Electrical stimulation of vagus in neck gave no reaction. Then 1 c.c. formalin was blown upon left visceral pleura. Soon a vasomotor type of fall in pressure developed and death ensued. Here the atropine abolished the "vagal" reflex, but not the vasomotor. Postmortem: Brain normal. Right pleura covered with thick layer of lymph. Left pleura moderately inflamed. Congestion of abdominal vessels.

Experiment 49. A double bilateral pleurisy followed forty-eight hours after introducing 200 c.c. of dirty oil in right thorax. After the removal of about 50 c.c. of exudate from the chest the blood pressure showed little change. The application of 0.5 c.c. of formalin to the right visceral pleura caused the long strokes with a mean fall in pressure of about 25 mm. Upon administering a milligram of atropine the long strokes quickly changed to short and more rapid stroke. This experiment indicates that the long strokes were due to excitation of the cardio-inhibitory branches of the vagus, since they disappeared when these fibers were paralyzed by atropine.

Experiment 50. A dog with a bilateral pleurisy, forty hours after the introduction of 200 c.c. of contaminated oil in the left pleural cavity. Initial blood pressure was 120 mm. After slow aspiration of over 100 c.c. of fluid the pressure was 100 mm. The injection of 1 c.c. of formalin on the left visceral pleura was followed by slow and shallow breathing and by a rapid fall in blood pressure to about 20 mm., with frequent long strokes. Immediate section of both vagus nerves in the neck caused a sudden rise in pressure to 75 mm., with increased rapidity of pulse, but the recovery was only transitory. The second fall was rapid and free from long strokes. Postmortem: About 50 c.c. of fluid in the left cavity and fibrin deposits. Right fibrinous pleurisy, with no free fluid. Engorgement of abdominal bloodvessels. We may infer that two reflexes conspired to depress blood pressure in this experiment; the one acting on the medullary centre through the vagus nerves and eliminated by section of the same; the other, of vasomotor type, causing vasodilatation of the abdominal vessels and not affected by section of the vagi.

Experiment 51. A dog had been injected in the right pleura over forty hours previous to experiment with 10 c.c. of contaminated oil. About 50 c.c. of exudate was aspirated without any influence on pressure. Then about 200 c.c. of a 1 per cent. formalin solution was forced into the pleural cavity, care being taken to avoid pneumothorax. The fluid was then allowed to flow out by siphonage. The irrigation was accompanied by marked slowing of respiration and a combined type of cardio-inhibitory and vasomotor pulse tracing. Respirations then ceased entirely, section of both vagi in the neck abolished the "vagal" strokes, but the pressure continued to fall until it reached about 10 mm. Hg.; then spontaneously breathing started up again and the blood pressure arose steadily. The animal recovered. (Fig. 6).

Summary of Experiments. Aspiration of oil from the pleural cavity of 6 healthy dogs caused only a slight lowering of blood pressure. Aspiration of oil from the chest of 7 dogs in which pleurisy had been produced caused in 3 little effect, in 3 a considerable fall in pressure and in 1 death.

Irritation of the visceral and parietal pleura in 9 healthy dogs produced no marked effect upon blood pressure.

In the group of 6 pleurisies produced by turpentine 16 per cent. gave a marked blood-pressure-lowering reflex in response to irritation of the trocar, while 50 per cent. gave such a reflex from formalin irritation.

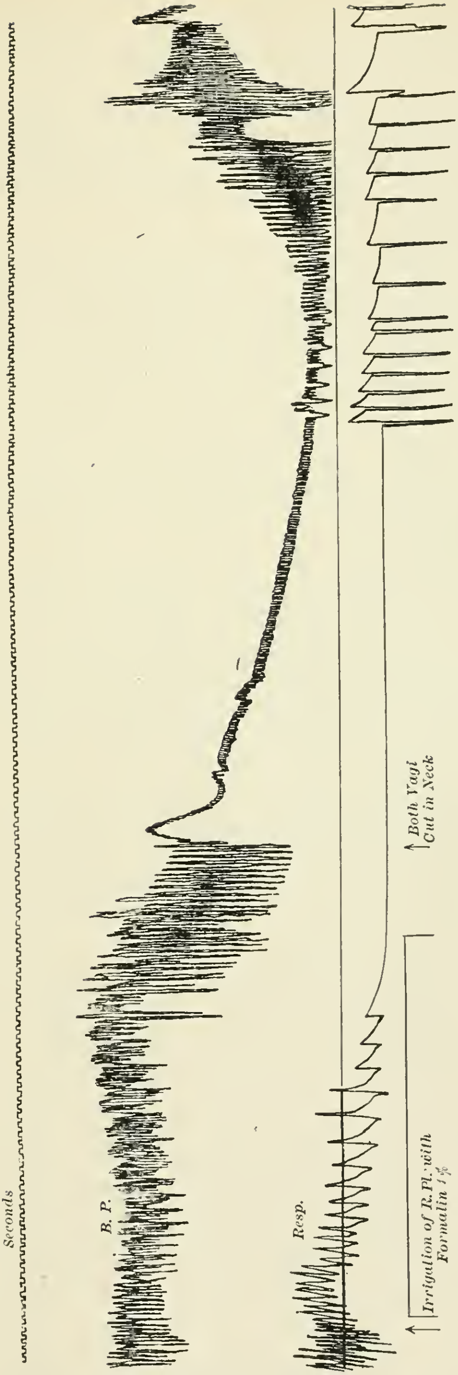

Fig. 6.—*Combined cardio-inhibitory* and *vasomotor reflex* resulting from irrigation with 1 per cent. formalin in glycerin of pleural cavity of dog with artificial empyema. At the top is the time record in seconds. Upper tracing represents blood pressure, the lower respirations, between them the base line. Respirations ceased at beginning of long strokes. Section of both vagi in the neck abolished the long "vagal" strokes, but the vasomotor fall continued. Respirations and blood pressure recovered spontaneously.

Of the group of 5 pleurisies caused by the injection of oil contaminated with bacteria and operated on within twenty-four hours after injection, 20 per cent. showed from the trocar irritation a decided blood-pressure-lowering reflex and 25 per cent. from formalin irritation.

In the group of 17 pleurisies induced by the presence of bacterially contaminated oil in the chest for forty-eight hours or more, 42 per cent. responded to the trocar irritation with a marked depressing reflex and 46 per cent. responded in like manner to the formalin.

Of the total 28 cases of pleurisy of all kinds, 33 per cent. gave a marked blood-pressure-lowering reflex from trocar irritation, while of a total of 17 cases in which formalin irritation was employed, 47 per cent. exhibited a decided fall in blood pressure.

Artificial respiration with the thorax opened tends to abolish all reflexes from irritation of the viscera, as was first pointed out by Brodie and Russell.

It is noteworthy that the trocar irritation seldom gave rise to this reflex in the turpentine pleurisies and in the bacterial pleurisies of short duration, but frequently caused it in the bacterial pleurisies of longer duration. The formalin,[6] on the other hand, induced the reflex in one-half of the turpentine pleurisies and with about the same frequency in bacterial pleurisies of both long and short duration.

Why the reflex occurred in some cases and was entirely lacking in others was explained in part only. In certain instances a very *thick fibrinous exudate* was found, which may have protected the pleura from injury and thus prevented the reflex. But in most cases this variability of reaction was not made clear by anatomical conditions.

The *location* of the irritation was of some importance. The nearer the irritant approached the root of the lung where the fibers of the pulmonary nerves spread out, the more likely was the reflex to occur. The fall in blood pressure arising from mechanical irritation was usually of the vasomotor type; the fall arising from formalin irritation was sometimes of the vasomotor, sometimes of the cardio-inhibitory type, and occasionally seemed to be a combination of both types. It may be noted that the cardio-inhibitory type, though often alarming in its symptoms, never alone caused the death of an animal. The vasomotor type of reflex, however, was always dangerous and several times fatal. Twice death was prevented by adrenalin injections.

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[6] Subsequent experiments indicate that when formalin is introduced in *large quantity*, say 2 to 20 c.c. in the pleural or peritoneal cavity of healthy dogs, a marked lowering of blood pressure often occurs, due in part to absorption in the circulation. We believe, however, that absorption plays a minor rôle when formalin is applied to the visceral pleura in small quantities.

Anatomical Course of the Reflexes. Before discussing the possible courses of these reflexes it seems advisable to review the innervation of the pleura and heart of the dog, and the anatomical arrangement and course of the fibers in the pneumogastric and sympathetic nerves.

The bronchial branches arising from the pneumogastric nerves are given off from the ventral and dorsal aspects of the nerve a little aboral to the aortic arch. The ventral branches pass along the large vessels and accompany the bronchi into the lungs, while the dorsal branches pass along the œsophagus, trachea, and lymph nodes to the root of the lung.

All these fibers unite to form at the root of the lung an extensive network, which is called the pulmonary plexus. The part of the plexus which usually lies upon the principal bronchus and more on the ventral surface is called the anterior pulmonary plexus, while the part lying aboral to the principal bronchus is called the posterior pulmonary plexus. These two plexuses are connected by branches, and the pulmonary is connected with the cardiac plexus.

The stellate ganglion marks the beginning of the thoracic part of the sympathetic ganglionated cord. It is oval and flattened, and lies lateral to the vertebræ upon the m. longus colli at the level of the second rib, and also extends downward to the second intercostal space. From this ganglion are distributed a number of branches, to the first and second thoracic nerves, the lower cervical nerves, and the two cardiac branches (rami accelerantes cordis), which assist in the formation of the cardiac plexus lying upon the lateral surface of the aortic arch and the base of the heart.

The cardiac plexus sends fibers into the pulmonary plexuses above described, from which branches are distributed along the vessels and bronchi to the pleura. Small sympathetic ganglia are interposed at frequent intervals along the nerves. Branches are given off from this plexus, of which the vagus also forms a part, that supply the pericardium and heart muscles. The pleura is innervated therefore by elements of both sympathetic and vagus nerves.

The vasoconstrictor nerves of the body leave the spinal cord by the anterior roots of the spinal nerves from the first dorsal to the third or fourth lumbar inclusive. From the roots they pass by the white rami communicantes to the ganglia of the sympathetic chain lying along the front of the vertebral column. Here they take different courses according to their destinations. The fibers for the head and neck leave by the first four thoracic nerves, pass into the sympathetic chain through the stellate ganglion and the ansa Vieussenii to the inferior cervical ganglion, and up the cervical sympathetic trunk to the superior cervical ganglion. Here they end, and the impulses are carried by a fresh relay of fibers, starting from cells in this ganglion and travel along non-medullated fibers on the walls of the carotid artery and its branches.

The most important vasomotor nerve is the splanchnic nerve. This nerve receives most of the fibers forming the white rami from the lower seven dorsal and upper two or three lumbar roots, the latter fibers often taking a separate course in the lesser splanchnics. The fibers can be seen to pass through the sympathetic chain of the thorax without interruption and for the most part have their cell station in the large ganglia, especially the semilunar ganglia of the solar plexus, whence a thick meshwork of non-medullated fibers is distributed along all the vessels of the abdominal viscera. The area of vessels innervated by this nerve is so large that section of this nerve on each side causes a large fall in general blood pressure.

THE CARDIO-INHIBITORY REFLEX.—The typical cardio-inhibitory reflex obtained by irritation of the inflamed pleura corresponds to that elicited by electrical stimulation of the vagus nerve in the neck. The blood-pressure tracing shows long downward strokes, with a marked difference between systolic and diastolic blood pressures, slowing or temporary arrest of the pulse, and frequently slowing of the respiration. Unless the current is very strong the heart overcomes the inhibitory action and the blood pressure returns to its original level. This reflex was never obtained in healthy dogs by trocar irritation of the pleura or of the thoracic sympathetic nerves. Long, single strokes without slowing of the heart were frequently obtained by scratching the root of the lung or the thoracic portion of the vagus nerve itself or by striking the pericardium with the trocar. These latter curves have very little effect on the mean blood pressure, and because of their slight importance were not recorded as pathological cardio-inhibitory reflexes.

We believe that the cardio-inhibitory reflex resulting from irritation of the inflamed pleura was usually of central origin, arising in the afferent branches of the vagus and travelling upward through the pulmonary ganglia to the combined nucleus of the vagus and accessory nerves in the medulla, the inhibition being transmitted along the accessory fibers to the vagus nerve back to the cardiac plexus and finally to the heart. (Fig. 7.) We believe the reflex to be of central origin, as it was usually absent after section of both vagi.

Atropine in the dosage of 1 mm. injected into a vein either diminished or prevented the reflex.

VASOMOTOR REFLEX. The vasomotor (dilator) reflex differs from the cardio-inhibitory in that the long "vagal" strokes are usually wanting and the mean fall in blood pressure is progressively downward, often resulting in death. The respiration may or may not be slowed. The nature of this reflex may be made clear by the following evidence:

1. Acute engorgement of the vessels of the abdominal viscera was found in all cases, evidently from acute vasodilatation.

2. Section of the vagi in the neck did not prevent the reflex or

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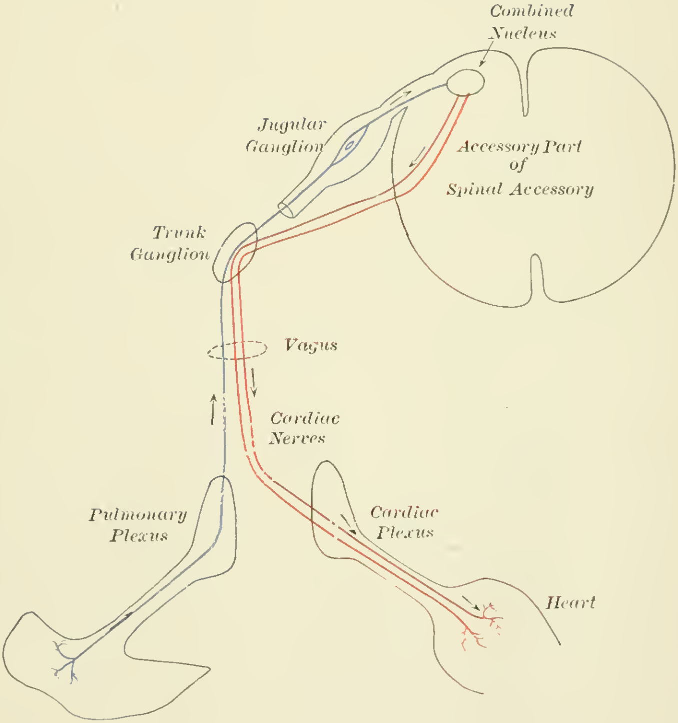

FIG. 7.—Illustrating the course of the cardio-inhibitory reflex. The impulse is carried by the afferent fibers (blue) from the visceral pleura, through the vagus, to the medulla; thence back to the heart by the efferent nerves (red).

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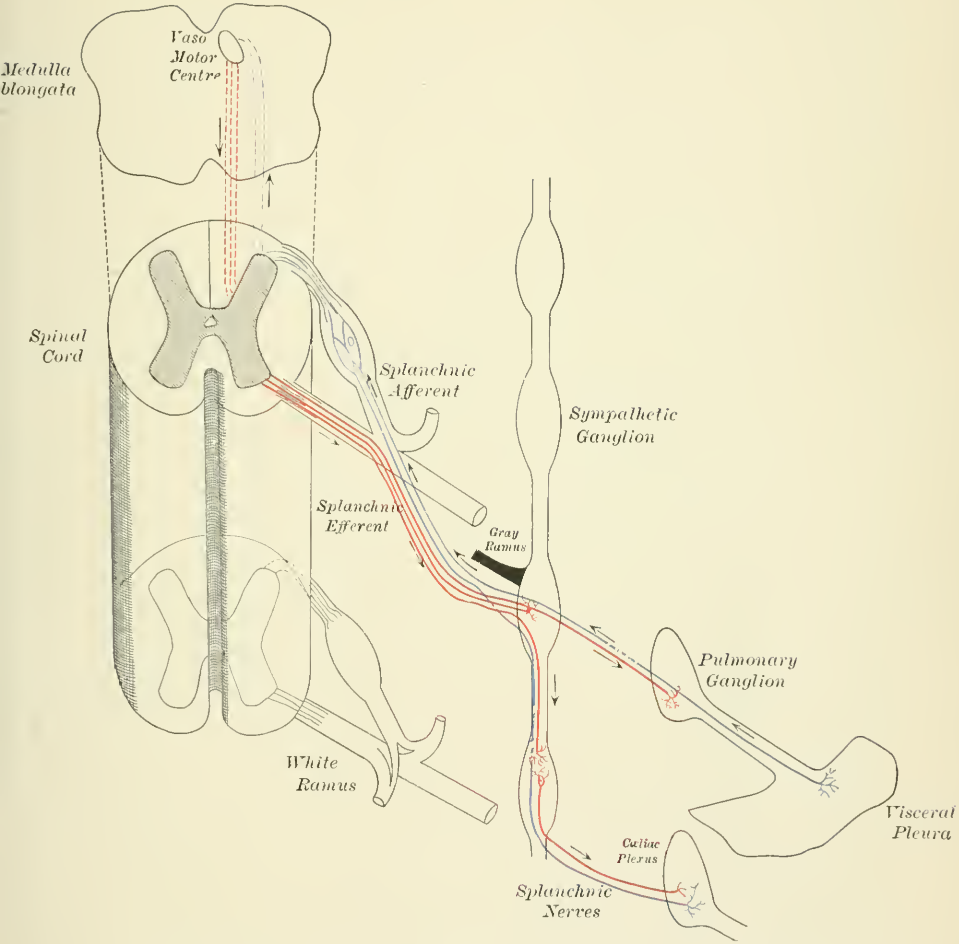

FIG. 8.—Illustrating the course of the vasomotor reflex. The impulse is conducted by the afferent nerves (blue) from the visceral pleura, through the pulmonary ganglia, the sympathetic cord, and the posterior roots to the vasomotor centre in the medulla. From there it is carried by the efferent nerves (red) along the anterior roots, sympathetic cord, and splanchnic nerves to the cœliac plexus.

alter it after it was induced, which indicates that the reflex was independent of any inhibitory action of the heart.

3. Intravenous injection of adrenalin, a vasoconstrictor, opposed the reflex, causing an immediate rise in blood pressure. Twice the injection of adrenalin solution saved the life of the animal. Atropine, on the other hand, which paralyzes the cardio-inhibitory fibers of the vagus, had no effect.

According to our present knowledge of the course and distribution of the nerves of the sympathetic system it seems probable that this reflex is central rather than peripheral, that is, that the afferent impulse is carried back to the principal vasomotor centre in the medulla or subsidiary centres in the cord, and that the efferent impulses are then transmitted along the white rami to the collateral ganglia. As the reflex is accompanied by such a marked fall in blood pressure it seems probable that the reflex occurs through the principal rather than the subsidiary centres.

The afferent fibers represented in blue (Fig. 8) probably traverse the sympathetic cord, passing upward, downward, and outward without arborizing about these cells in the ganglion and, therefore, without losing their medullary sheath. It is quite generally accepted now that the afferent fibers which are sensory have this arrangement. A reflex beginning in the pleura will traverse these fibers, passing through the posterior root, and the reflex may be established at the level at which the fibers enter the cord, or they may pass upward to arborize about the cells in the principal vasomotor centre in the medulla. This anatomical arrangement precludes the possibility of a more direct reflex arising from the pulmonary plexus and travelling directly along the thoracic ganglionated cord to the splanchnic vessels.

It should be mentioned, however, that Dogiel[7] has described a different arrangement of the fibers in the sympathetic cord, and if his interpretation is correct a peripheral reflex arising in the peripheral plexuses and traversing directly the ganglionated cord is possible.

We have already noted that the effects of this reflex are so general and rapid that it seems probable that the principal vasomotor centre is affected and not the subsidiary centres situated in the cord.

CLINICAL OBSERVATIONS IN MAN. The following observations were made in man to ascertain if irritation of the inflamed pleura during thoracentesis produced any marked changes in blood pressure:

CASE I.—The patient had a pleurisy with effusion of over two weeks' duration. Thoracentesis in midaxillary line, sixth space. Fluid was seropurulent and contained pneumococci. The initial blood pressure was 100 mm. Hg. After withdrawal of 125 c.c. of fluid the flow ceased. Blood pressure, 98 mm. The trocar was then moved about in the endeavor to start the flow. The visceral pleura was distinctly felt against the trocar during these movements. The blood pressure rapidly fell to 80 mm. when the instrument was withdrawn. In four minutes the blood pressure had risen again to 96 mm.

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[7] Poirier et Charpey, Traité d'anatomie humaine.

Case II.—Man with a right-sided streptococcic empyema of over a month's duration. Needle inserted near angle of scapula. Initial blood pressure 105 mm. Hg.; after entrance of needle, 98 mm. As no fluid escaped, the needle was moved about and the pleura intentionally scratched with the end. The blood pressure fell immediately to 88 mm. and after a short rest recovered to 94 mm. A few more scratching movements caused a rapid fall to 84 mm. with intermissions of the pulse. Exploratory puncture was then made in the midaxillary line, in the sixth space. Blood pressure after entrance of needle, 90 mm. No fluid was obtained. A few lateral movements of the needle were made against the visceral pleura, when the systolic blood pressure fell to 75 mm., the diastolic pressure below 35 mm., and for a few seconds the pulse could not be felt. Patient felt slight dizziness and faintness. Upon withdrawal of needle the pulse rapidly improved and the subjective symptoms disappeared.

Case III.—Right pleurisy with bloody effusion. At postmortem a month later, the lung was found retracted and tied down with tuberculous adhesions. Trocar in sixth space, midaxillary line. Initial blood pressure 89 mm. Hg. After insertion of trocar 79 mm. 1200 c.c. of fluid was withdrawn by siphonage without discomfort. Blood pressure, 69 mm. Then the trocar came in contact with the visceral pleura and, although very little more fluid escaped, the blood pressure fell in a few minutes to 57 mm. and the patient complained of faintness, dizziness, and a dull pain at the site of the needle. The pulse beats were very difficult to feel and at times were intermittent. The trocar was removed and in three minutes the blood pressure was 62 mm. Lying down increased blood pressure to 67 mm. Thirty minutes later blood pressure was 80 mm. and the patient was comfortable.

Case IV.—Left pleurisy of ten days' duration. Thoracentesis in postaxillary line, seventh space. During the withdrawal of 1500 c.c. of fluid by slow siphonage no pain was experienced, and the blood pressure fell from 106 mm. to 100 mm. Scratching the visceral pleura gently with a needle was followed by only slight pain and a fall in blood pressure to 90 mm. In four minutes after needle was removed the blood pressure was up again to 100 mm.

Cases. V, VI, and VII were all acute pleurisies with serous effusions. In all cases there was very little reflex fall of blood pressure from irritating the pleura with the trocar.

Case VIII.[8]—Man of thirty years with left pleurisy and tuberculosis of the left lung. Thoracentesis in sixth space midaxillary line. Initial blood pressure 120 mm. Hg. After insertion of needle 130 mm. 2000 c.c. of serous fluid aspirated in nine minutes, when blood pressure was 105 mm. The needle end then came suddenly in contact with the visceral pleura and patient complained of dull pain in the epigastrium, but none whatever in the chest. The pain grew rapidly worse and the blood pressure rose steadily to 155 mm. —an increase of 50 mm. in the course of five minutes. Upon withdrawal of the needle the epigastric pain gradually subsided and the blood pressure simultaneously was lowered. Twelve minutes after the maximum anginal pain the blood pressure had returned to 108 mm. and the pain was gone. There is every reason to believe that excitation of the visceral pleura in this instance caused a reflex *vasoconstrictor* action on the abdominal arteries.

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[8] This case was published by Capps, loc. cit.

These observations go to show that in man, as in dogs, irritation of the inflamed pleura often gives rise to a blood-pressure-lowering reflex of cardio-inhibitory or vasodilator type. Also that similar excitation under similar conditions may give rise to a reflex of vasoconstrictor type, with heightening of blood pressure. This latter type of reflex is exceptional and has been observed only in this 1 case out of a total of over 30 patients in whom careful blood-pressure readings have been taken during the progress of aspiration. In our experiments on the pleurisies of animals we have never seen such a striking vasoconstrictor reflex, but several times a gradual rise in blood pressure has followed pleural irritation, which may possibly be explained in this manner.

ILLUSTRATIVE CASES IN LITERATURE. It does not lie within the scope of this paper to discuss the numerous cases recorded in literature of collapse or sudden death occurring in the course of operations in the thorax in which no satisfactory cause could be found postmortem. But it may be pertinent to allude briefly to a few writers who have helped to elucidate the phenomenon.

Sears[9] has recently published a full bibliography besides reporting a case in which death followed fifteen hours after exploratory puncture. Russell[10] described 3 cases, 2 of them children, that died after exploratory puncture and gives the most instructive discussion of the subject at our command. As previously stated he considers that in these cases the needle penetrates the lung tissue and excites vagus nerve endings in the alveoli. He believes that the pleura takes no active part in the reflex—a conclusion entirely opposed to our own. Leichtenstern[11] reports three groups of fatal cases that occurred from operative procedures: 4 patients died suddenly during aspiration; 15 patients died a few minutes to a few days after operation; 5 patients died during irrigation of the pleural cavity, some with partial or general convulsions. Rosenbach[12] reported a sudden death from irrigation of the pleural cavity in a patient with empyema, in which the postmortem findings were entirely negative. He attributed the probable cause of death to irritation of the vagus-nerve endings. He alludes also to an interesting historical case of sudden death resulting from the accidental loss of a part of a syringe in the pleural cavity following an operation for empyema. Auberne[13] describes convulsive attacks accompanying irrigations of the pleura which are sometimes followed by syncope and death, at other times by recovery after a short interval without any bad consequences. Weill[14] refers to similar convulsive movements during irrigation of the chest, especially on the affected side. The convulsions are usually explained by an anemia of the brain which accompanies the reflex fall in the blood pressure (Russell, Rosenbach).

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[9] Trans. Assoc. of Amer. Phys., 1906.

[10] Loc. cit.

[11] Deutsch. Arch. f. klin. Med., 1879, xxv.

[12] Loc. cit.

From these illustrations it is evident that the pleural reflex lowering blood pressure may be caused by a number of forms of irritation of the pleura: (*a*) by irritation or injury of the needle, (*b*) by a foreign body in the pleural cavity; or (*c*) by irrigations of the cavity with antiseptic solutions. Obviously if the susceptibility of the inflamed pleura to dangerous reflexes were fully appreciated, greater care would be used by physicians and surgeons to avoid any unnecessary injury or irritation of the membrane during operations and irrigations.

CONCLUSIONS. 1. Aspiration of oil from the pleural cavity of healthy dogs causes little or no change in the arterial pressure.

2. Aspiration of inflammatory exudate from the pleural cavity of dogs with acute pleurisy often causes a more marked fall in blood pressure. This fall in pressure depends more on the degree of trauma or irritation of the inflamed pleura than on the amount of exudate withdrawn or the rate of withdrawal.

3. Irritation of the visceral pleura of healthy dogs by mechanical, thermal, and electrical means and by certain chemicals, produces little or no effect on blood pressure, except over the roots of the lungs, where mechanical and electrical excitations produce long strokes of vagal type.

4. The effect of irritation of the parietal pleura needs further investigation. The common drop of blood pressure and disturbance of respiration occurring when the trocar is forced through the chest wall into the cavity is probably due to injury of the parietal pleura. The reflex is usually transitory and is seen in both normal animals and in those with pleurisy.

5. In dogs with pleurisy induced by turpentine or by oil contaminated with bacteria, excitation of the inflamed visceral pleura by mechanical and chemical irritants gives varying results. In some cases there is no marked change in blood pressure; in others there is a considerable fall in pressure that may even be fatal.

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[13] Revue de méd. et chir., February, 1879.

[14] Ibid., January, 1887.

6. These reflexes conform to two types, which as a rule occur singly, but may be combined: (*a*) The cardio-inhibitory type, in which the heart is slowed and the pulse tracings make violent excursions with a great range between systolic and diastolic pressure. Respirations also are usually slowed and may be inhibited. This type of reflex when it occurs alone is seldom fatal. (*b*) The vasomotor type, in which the pulse tracings show a steady rapid decline of pressure without a great difference in systolic and diastolic pressure and frequently terminate in death. Respirations as a rule are shallow and may be rapid. In fatal cases the bloodvessels of the abdominal viscera are much engorged from acute vasodilatation. The brain shows no evidence of embolism or hemorrhage.

7. The cardio-inhibitory reflex is central, because it is prevented or stopped by cutting both vagus nerves in the neck. Atropine in a dosage of 1 milligram paralyzes the cardio-inhibitory fibers and destroys the reflex.

8. The vasomotor (dilator) reflex may be central or peripheral. If central, the afferent impulses reach the medulla by way of the thoracic sympathetic, the white rami, and the cord, and not by the vagosympathetic cord. This is proved by the failure of section of the vagosympathetic cord to alter or abolish the reflex. If peripheral, the reflex goes from the pulmonary fibers to the pulmonary plexus, thence to the thoracic sympathetic nerves and downward through the splanchnics to the celiac and other plexuses in the abdomen. This reflex is more direct than the central form, but seems to us inconsistent with the views generally accepted as to the course and direction of impulses in the sympathetic nerves. Adrenalin is the physiological antagonist to the vasodilator reflex and is often life saving. Atropine on the other hand, by its tendency to dilate the cutaneous vessels and lower the mean arterial pressure, seems to intensify the reflex. There is some evidence that the previous administration of atropine modifies the blood pressure—elevating action of adrenalin and thereby deprives it of its full beneficial effect.

9. These types of reflexes occur also in man during operative procedures upon the inflamed pleura. The cardio-inhibitory type is manifested by a slow intermittent pulse, with a great difference between systolic and diastolic pressures; the vasomotor type by a steady fall in blood pressure, without a marked difference in systolic and diastolic pressures, and by a pulse that grows steadily weaker until it cannot be felt.

10. For emergency use in case of falling blood pressure and symptoms of collapse adrenalin intravenously is indicated. Atropine is of little service and may even do harm.

11. Preventive measures come readily to mind. The instrument used in thoracentesis should not irritate the visceral pleura any more than is absolutely necessary. Therefore, the trocar is preferable

to the needle. The trocar should not be inserted at a greater depth than is necessary to obtain fluid. Great care should be employed during the drainage of an empyema, especially to avoid a long projection of the drainage tube inside the cavity. Swabbing the pleural surface is attended with danger. Finally we would emphasize the clinical importance of taking blood-pressure readings at the beginning of and during all operations in the pleural cavity, in order to foresee and thereby prevent the development of a dangerous blood-pressure lowering reflex.

We gratefully acknowledge our indebtedness to Dr. S. A. Mathews, the director of the laboratory, for his constant assistance and encouragement.

REVIEWS.

The Principles and Practice of Medicine. By Arthur R Edwards, A.M., M.D., Professor of the Principles and Practice of Medicine and of Clinical Medicine in the Northwestern University Medical School, Chicago, Illinois. Pp. 1328; 101 engravings, and 19 plates. Philadelphia and New York: Lea Bros. & Co., 1907.

Dr. Edwards has prepared a *Practice of Medicine* which, although it follows familiar lines in many respects (and this from the nature of the subject can scarcely be avoided), yet possesses a sufficiency of individuality to arrest attention. The classifications adopted are those usually followed, and the treatment of much of the subject matter is very much what one expects. But, as the author states in the preface (and his statements are confirmed by a perusal of the book), as far as possible, the causative pathology has been blended with the consecutive clinical features of disease, reasons have been given for facts, exceptions have been subordinated to what is usually found at the bedside, and the allurements of typical clinical pictures and dogmatic generalizations have been avoided because they hold neither in practice nor at the postmortem table. The pathological and clinical descriptions are clear, concise, and accurate: this is especially true of many of the infectious diseases and diseases of the nervous system. But, here and there, especially in the sections devoted to diseases of the heart and of the gastro-intestinal system, the style adopted does not lend itself well to an orderly description of the evolution of the diseases in question—which is especially important from the point of view of the student. There is a large number of excellent tables illustrating the differential diagnosis of diseases likely to be confused, and tables of diseases of certain organs, such as the liver and the kidneys, and of the eruptive diseases and diseases of the typhoid group. In these and in general in the paragraphs devoted to differential diagnosis there is often a wealth of facts not to be found in other works of a similar kind. An unusual amount of space has been devoted to treatment, to the detailed consideration of drugs, and to numerous formulæ and prescriptions ready, as the author states, for the student to use or improve upon. The physiological action of drugs has been dwelt upon because, in the author's experience, the symptoms of disease are often confounded with those of the remedies exhibited for its cure. This is an excellent feature of the book—wisely undertaken and well executed. Considerable attention also has been devoted to the indications for and the results of surgical intervention in many of the so-called borderland diseases.

As a whole, the book impresses the reviewer very favorably; he recommends it to the attention of students, practitioners, and teachers, in the belief that when tried it will not be found wanting, but will be always of service. A. K.

GYNECOLOGY AND ABDOMINAL SURGERY. Edited by HOWARD A. KELLY, M.D., F.R.C.S., Professor of Gynecological Surgery in the Johns Hopkins University; and CHARLES P. NOBLE, M.D., Clinical Professor of Gynecology in the Woman's Medical College of Pennsylvania, Philadelphia. Vol. I; pp. 851; 405 original illustrations by Hermann Becker, Max Brödel, and others. Philadelphia and London: W. B. Saunders Co., 1907.

WE have looked forward with eager pleasure to the publication of the present work from the early stages of its preparation, and therefore it gives us an added measure of satisfaction to be able to say that careful reading of this first volume has not left us with a sense of disappointment. It is needless to say that the work has been thoroughly done: the names of the authors and editors would guarantee this, but much may be said in praise of the method of presentation, and attention may be called to the inclusion of matter not to be found elsewhere. We are greatly pleased with the character of the illustrations prepared by Messrs. Becker and Brödel, and we would particularly call attention to the insertion of the bibliography at the foot of the page. This is a German method which has but too few imitators in this country. Every book which makes any pretensions to authority should give ample space to the citation of references to the original sources of information, and it gives us great pleasure to note this admirable feature of this book. In addition we would call especial attention to the historical *resume* which precedes the consideration of each subject. The history of medicine is but too little considered in the rush of present-day work, and we therefore welcome most heartily this variation from the usual.

Three of the authors of the volume have died since the inception of the work, Drs. Skene, Pryor, and Heurotin, and their articles must have an added interest in that they are the last contributions of these well-known men to surgery. Considering the book from the standpoint of technical criticism, there is but little to regret and much to praise. Of course, it would be possible to find some typographical errors by careful search; probably there might have been more illustrations inserted in certain portions and fewer in others, or other equally valueless criticisms which might pass as an erudite review might be made, but if one reads the book with a remembrance that the authors make no claim to superhuman attributes we feel certain that the verdict will be unqualifiedly favorable. The editors, Drs. Kelly and Noble, have been most happy in the choice of their collaborators; the names of Ford, Clark, Baldy, Hurd, Fullerton, Werder, Henrotin, Hurdon Edebohls, Anspach, Hunner, Skene, McMonagle, Byford, and Webster, with the editors, Drs. Kelly and Noble, are sufficient assurance to the man versed in the gynecological literature of the present day of the excellence of the work. A detailed description of the contents of the volume is impossible because of lack of space, but we would call attention to the chapter by Kelly upon gynecological technique, to Ford's discussion of bacteriology, to Hurdon's presentation of the pathology of the reproductive organs, which chapter appeals to us as the best we have ever read, and to the elaborate chapter by Noble and Anspach upon medical gynecology. This last-mentioned portion of the book is one of the landmarks of the work, its inclusion reflecting as great credit upon the sagacity of the editors as does its excellence upon the ability of the authors. It is unique in scope, covering more than one hundred pages. The chapter by Edebohls upon the history of the development of the combined gynecological operation is of great interest, and Hunner's chapter upon the bladder and urethra is particularly well done. Baldy's chapter, upon hysterectomy in the presence of inflammatory disease of the uterine appendages, is characteristically clear and concise, while Noble's abdominal hysteromyomectomy and myomectomy has the charm which attaches to the work of the enthusiast. We are particularly pleased to note his stringent limitations of the latter procedure. Fullerton and Noble divide the operative procedures upon the external genitalia, the former considering the nonplastic work, while the latter devotes his attention to the repair of the injuries incident to childbirth, etc. A special section is devoted to ventrosuspension by McMonagle. To Skene has been assigned ovariotomy, including the clinical history and diagnosis of ovarian tumors, while vaginal section for disease of the ovaries and tubes is described by Byford. The chapter by Webster upon the abdominal removal of these organs completes a most satisfactory study of this whole subject. Clark considers the radical operation by the abdominal route in the presence of cancer of the uterus. An interesting historical *resume* is given, the illustrations are especially good, and, while his conclusions may be differed from in part, the fairness of his presentation is most gratifying. We are glad that just at this time and in a book of this character it has seemed good to include a description of the Byrne method of attack. Werder makes the chapter extremely interesting and his modification of the original plan is well worth study and consideration.

The last three chapters, upon vaginal hysterectomy by Henrotin, conservative operations upon the tubes and ovaries by Kelly, and operations before puberty by Kelly and Hurdon, are all pleasing, the first mentioned because of its clearness and attention to detail, the second because of the fairness of its presentation, and the third because of its uniqueness, the data herein presented being otherwise available only after laborious search through various original articles.

We await the second volume with the greatest interest, dealing as it will with the general surgery of the abdomen. We feel that the editors are to be congratulated upon the publication of the work just at this time, when the old, narrow limitations of gynecology, as distinct from general abdominal surgery, are rapidly ceasing to exist.

W. R. N.

CLINICAL DIAGNOSIS BY MEANS OF MICROSCOPIC AND CLINICAL METHODS. By CHARLES E. SIMON, B.A., M.D., Professor of Clinical Pathology in the Baltimore Medical College, Baltimore, Maryland. Sixth edition; pp. 682; 177 engravings, and 24 plates. Philadelphia and New York: Lea Bros. & Co., 1907.

THE sixth edition of Simon's *Clinical Diagnosis* comes to us as an old and tried friend well endowed with modern attainments. The book has been revised throughout. All obsolete matter has been eliminated, and by omitting also everything not of practical value room has been found for the inclusion of much new material. The chapter on the blood has been enlarged and brought thoroughly up to date; a new chapter on the opsonins has been introduced, and two appendices have been added—the first dealing with the preparation of culture media, and the second representing an outline of a course in clinical laboratory methods prepared at the request of many teachers of clinical microscopy. In its new edition the book remains, as it long has been, undoubtedly the best of its kind in the English language.

A. K.

THE ESSENTIALS OF HISTOLOGY. By A. E. SCHÄFER, LL.D., Sc.D., F.R.S., Professor of Physiology in the University of Edinburgh. Seventh edition; pp. 507; 553 illustrations. Philadelphia and New York: Lea Brothers & Co., 1907.

SCHAFER'S *Essentials of Histology*, now in its seventh edition, is widely recognized as one of the really excellent books on the subject. With each succeeding edition it has been modified and improved in accordance with the advances in our knowledge of microscopic anatomy. In the latest edition, the especially noteworthy change consists in considerable amplification of the description of the structure of the nervous system; but many and important additions are to be found elsewhere throughout the book. One of its many excellent features is the division of the subject matter into fifty lessons—which provides a well-arranged and systematic course for students; another is the numerous diagrammatic and other illustrations. The book is to be much commended, especially since it aims at giving only the essential facts, unimportant details being omitted; and doubtless it will continue to enjoy the favor of teachers and students of histology. A. K.

Practical Fever Nursing. By Edward C. Register, M.D., Professor of the Practice of Medicine in the North Carolina Medical College. Pp. 352. Philadelphia and London: W. B. Saunders Co., 1907.

Nursing in the Acute Infectious Diseases. By George P. Paul, M.D., Assistant Visiting Physician to the Samaritan Hospital, Troy, New York. Pp. 200. Philadelphia and London: W. B. Saunders Co., 1907.

Materia Medica for Nurses, Including Therapeutics and Toxicology. By George P. Paul, M.D., Assistant Physician to the Samaritan Hospital, Troy, New York. Pp. 242. Philadelphia and London: W. B. Saunders Co., 1907.

Materia Medica for Nurses. By Emily A. M. Stoney, late Head Nurse, Mercy Hospital, Chicago, Illinois. Third edition. Pp. 300. Philadelphia and London: W. B. Saunders Co., 1906.

Obstetrics for Nurses. By Joseph B. De Lee, A.M., M.D., Professor of Obstetrics in the Northwestern University Medical School, Chicago, Illinois. Second edition. Pp. 510. Philadelphia and London: W. B. Saunders Co., 1907.

The foregoing form an excellent series of books for nurses. Register's *Practical Fever Nursing* is in many respects one of the best books on the subject with which we are acquainted, although perhaps it errs in presenting more of pathology, symptomatology, diagnosis, and treatment than reasonably should be expected of a nurse; but Dr. Register states his plan has been followed because of his belief that a nurse cannot know the cause and significance of many of the symptoms unless she knows something of the pathological processes that are going on within the body, nor can she anticipate all that is expected of her by the physician unless she is at least partly familiar with the history and treatment of the fever which she is nursing. There is, doubtless, a modicum of truth in this, but there is a special danger attendant upon the use of such books—that pertaining to all superficial and misdirected knowledge. Apart from some tendency in this direction, Dr. Register's book is excellent, especially the chapters on general considerations and prophylaxis.

Paul's *Nursing in the Acute Infectious Diseases* is a much smaller book, which, however, contains the essentials of the subject. The author discusses first the fever process and nursing in general; then special nursing in the different infectious processes; and finally certain related subjects of value in connection with caring for fever patients. The book is excellently adapted to serve as a first book on the subject in training schools, and might well be followed by the use of Register's.

Paul's *Materia Medica for Nurses* is divided into six parts—general considerations (drug constituents, preparations, etc.); general materia medica, therapeutics, and toxicology; drugs of minor importance; newer medicinal agents; practical therapeutics; and certain addenda (weights and measures, etc.). The book is excellent in every way—the author having struck the happy medium between too much and too little.

Stoney's *Materia Medica for Nurses*, now in its third edition, has been well tried and much commended. As is well known, it comprises an excellent discussion of familiar topics, and it has been revised in accordance with the new Pharmacopœia.

DeLee's *Obstetrics for Nurses* is an unusually good book, containing perhaps a little too much of purely medical information more suitable for the junior medical student than for the nurse, but assuredly well adapted to the purposes in mind. The author has succeeded well in combining obstetrics for nurses with obstetric nursing, so well, indeed, that young physicians might read the book with profit. The book is exceedingly well illustrated, largely from photographs of actual obstetric scenes. A glossary of obstetric terms adds much to the value of the volume.

A. K.

TROPICAL MEDICINE, WITH SPECIAL REFERENCE TO THE WEST INDIES, CENTRAL AMERICA, HAWAII, AND THE PHILIPPINES, INCLUDING A GENERAL CONSIDERATION OF TROPICAL HYGIENE. BY THOMAS W. JACKSON, M.D., Lecturer on Tropical Medicine, Jefferson Medical College, Philadelphia, etc. Pp. 536; 106 illustrations. Philadelphia: P. Blakiston's Son & Co., 1907.

THE author states that his purpose has been to prepare for American medical men and students a simple and systematic presentation of the known and determined facts concerning such tropical diseases as are found within the boundaries of our own country, and that the guiding ideas that have determined the size, scope, and character of the book have been utility and simplicity. The need of such a book is obvious, since, during recent years, tropical medicine has enlisted the interest not only of those public, medical officials whose duty brings them in contact with disease is our tropical possessions, but also of many civilian practitioners, of inland towns as well as seaports, who with increasing frequency are called upon to diagnosticate and treat imported tropical infections. How well the author has attained his object will be apparent to all who read the book. It begins with an excellent chapter on tropical hygiene, in which due attention is paid to divers causative factors, including the influence of mosquitoes and other insects, and measures of prophylaxis. Then follows a systematical discussion of diseases prevalent in the tropics: (1) Systemic diseases, such as cholera, beriberi, plague, dengue, dysentery, leprosy, malaria, Malta fever, and yellow fever; (2) animal parasitic diseases, such as ankylostomiasis, filariasis, trypanosomiasis, bilharzia disease and schistosomum japonicum, endemic hemoptysis, Guinea worm disease, liver and intestinal fluke worms, and intestinal cestodes and nematodes; and (3) diseases of undetermined causation, such as acute febrile icterus, epidemic dropsy, yaws, climatic bubo, etc. The descriptions of these diseases in general is quite accurate and satisfying, and reference is made to the latest results of researches in the field of etiology. Much that is interesting and instructive is more or less personal: for instance, the author's experience with imported cholera at a station thirty miles from Manila; the satisfactory results of immunization against cholera on the transport Sherman; reference to outbreaks of malaria at sea; the occurrence of afebrile malaria, etc. A very useful feature of the book consists of detailed instructions for the laboratory diagnosis of many of the diseases—appended at the end of the respective chapters. The book is really of much value and contains a considerable amount of useful information—well arranged and readable. A. K.

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY. By W. A. NEWMAN DORLAND, A.M., M.D., Assistant Obstetrician to the Hospital of the University of Pennsylvania. Fourth edition; pp. 836. Philadelphia and London: W. B. Saunders Co., 1906.

THE AMERICAN POCKET MEDICAL DICTIONARY. Edited by W. A. NEWMAN DORLAND, A.M., M.D., Assistant Obstetrician to the Hospital of the University of Pennsylvania. Fifth edition; pp. 578. Philadelphia and London: W. B. Saunders Co., 1906.

BOTH of these dictionaries, from the time of their original publication, have enjoyed an enviable reputation. From time to time, as the progress of the medical sciences required it, they have been carefully revised, so that we find them now, in their latest editions, fully representative of the present state of medicine. A large number of new words—in the larger dictionary as many as 2000, and many of them of the latest coinage—have been introduced. To the larger dictionary six new colored plates have been added—illustrating appendicitis, diphtheria, gallstones, Leishman-Donovan bodies, measles, and nephritis. The definitions in both books are excellent—clear, concise, and accurate, but there is some little inconsistency in the use of *c* and of *k* in spelling words derived from the same Greek root. Both dictionaries may be cordially recommended—the smaller to the junior student, the larger to the senior student and practitioner—as accurate, complete, and fully abreast of the present state of medicine and the allied sciences. A. K.

A Manual of Diseases of the Nose, Throat, and Ear. By Edward B. Gleason, M.D., LL.D., Clinical Professor of Otology in the Medico-Chirurgical College, Philadelphia. Pp. 556. Philadelphia and London: W. B. Saunders Co., 1907.

This book has been written, as stated in the preface, to supply the needs of students and general practitioners who desire the essential facts of laryngology and otology presented to them in as concise a manner as possible. The author's concise method of statement lends itself well to this object and his many years of experience as a teacher have acquainted him with his readers' needs. As might be expected, the book contains nothing strikingly original, nor does it vary greatly from the numerous other text-books on the subjects whereof it treats. It does, however, contain up-to-date descriptions of all the most recent advances in rhinolgy and otology, such as paraffin prothesis and the various operative procedures upon the sinuses. The recent submucous operations for the correction of septal deformities are thoroughly described. The author gives in detail his own method for the correction of deflection of the septum, which has proved of great value in the hands of many other operators, For the student or general practitioner who wishes a reliable guide at the outset of his studies in laryngology and otology there are few books which can be more heartily commended than this of Gleason's. F. R. P.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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REGIUS PROFESSOR OF MEDICINE, OXFORD UNIVERSITY, ENGLAND,

AND

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The Value of the Fermentation Test for Sugar in the Urine.—H. A. Christian (*Boston Med. and Surg. Jour.*, 1907, clvii, 178) discusses the fermentation specific gravity test for quantitative sugar estimations in diabetic urine. Roberts in 1861 described a method of determining the presence of glucose in diabetic urine by means of the decrease in its specific gravity following the action of yeast. In comparing his results with the other methods in vogue at this time he was convinced of their accuracy. Of late this method has been neglected for other quantitative tests, such as titration with some form of copper sulphate, polariscopic examinations, and fermentation methods. The fermentation test has been compared with Fehling's and the polariscopic methods during the last two years at the Massachusetts General Hospital, and the results, graphically charted, show how closely the readings of the three methods agree. The procedure is as follows: Take the specific gravity of the urine at the room temperature; add a small bit of commercial yeast and place in a warm spot such as an incubator at 37° C., or in a heated room near a stove or radiator. When the bubbles of gas cease to appear, the urine should be tested with Fehling's or Nylander's solution, a negative result showing that the fermentation is at an end (usually twelve to thirteen hours). The specimen is then allowed to return in the room to the temperature of the previous specific gravity determination, and the specific gravity is redetermined. The difference between these two readings multiplied by 0.23 gives the percentage of fermenting substance expressed in terms of glucose. Filtering the urine before the second specific gravity determination gave no appreciable difference. Ordinary good commercial specific gravity bulbs were used and their results compared with more accurate ones; calculating the necessary allowance for temperature changes, the differences between the two were very slight. The same bulb, however, should be used for each determination of specific gravity, as there are some differences between the bulbs themselves. The specimen of urine should be approximately at the same temperature at each reading, or specimens with and without yeast may be kept side by side for controls and compared as regards their specific gravity. Christian says that observing these few precautions, results as accurate as with the other methods may be obtained, while its simplicity and cheapness should recommend it to more general use.

The Presence of Typhoid Bacilli in the Lice of Patients Suffering from Typhoid Fever. ABE (from the clinic of Professor Matsushiti in Kioto, *Münch. med. Woch.*, 1907, liv, 1924) calls attention to the influence of parasitic insects in the spread of various infectious diseases. It is a well-known fact that the mosquito is a direct transmitter of malaria; flies often infect food with the bacilli of cholera and dysentery. Typhoid bacilli, as is well known, are present in the circulating blood in the majority of instances of typhoid fever. It is, therefore, easily conceivable that the various human parasites—lice, fleas, and mosquitoes—when they bite human beings affected with typhoid fever, contain the parasites. Pflügge and others have long been under the impression that such parasites might, under circumstances, transfer the infection. Tsujitani and Herzog have each cultivated the bacilli of plague with fleas which have bitten mice.

Abe collected lice and fleas from individuals infected with typhoid fever, soaked them for several minutes in a sterilized test tube in 0.1 per cent. corrosive sublimate solution and then, after having thoroughly washed them with sterile water, ground them up in a sterile mortar. This powder was inoculated under the skin of a white mouse and onto the culture media of Drigalski, Conradi, and Endo. The test tube which was used for the collection of the parasites was filled with 10 cm. of bouillon and placed for twenty-four hours in a thermostat at 37°. With this bouillon smear cultures were also made upon the above-mentioned typhoid media. The bacteria which grew from the typhoid-like colonies and in the internal organs of the animals in whom the injections were made were carefully tested by all modern methods. The results showed that the lice from the body and clothes of typhoid patients contained typhoid bacilli in three out of four cases; while fleas obtained from the nurses of typhoid patients in two instances failed to show bacilli. This latter result may depend upon the fact that but few fleas were obtained. The author concludes that the skin parasites of human beings "play, probably, an important role in the spread of the contagious diseases, especially in typhoid fever." [But the discovery that fleas and lice which have bitten typhoid fever patients contain typhoid bacilli is by no means proof that they are capable of conveying the disease by subsequent feeding upon a healthy individual.—W. S. T.]

Observations on Metabolism in Exophthalmic Goitre. SCORDO and FRANCHINI (*Il Policlinico*, 1907, xiv, 285) report some studies made in Grocco's clinic on the metabolism in exophthalmic goitre. Their observations lead them to the conclusion which has been reached by many others, that emaciation with a relative loss of fat and albumin is by no means the absolute rule in this disease, nor is it dependent upon the course of the clinical symptoms, as one may observe an increase of weight without manifest change in the general appearances. During the period of observation the two patients studied gained slightly in weight. They ascribe a nitrogen retention, observed in these cases, to the fact that the patients were steadily improving, were at rest, and, in one case at least, were taking abundant nourishment.

There was a slight diminution in the oxidative processes as revealed by the diminution in the amount of nitrogen in urea as compared to the total nitrogen. There was an increase in amido acids dependent apparently on altered conditions of the hepatic circulation in both patients. There was a slight increase in uric acid and in the nitrogen precipitated by the phosphotungstic chloride mixture. There was an increase in the earthy phosphates and a marked increase in the elimination by the feces of P_2O_5, accounted for probably by the influence of the altered thyroid function as well as by the marked increase in the excretion of lime in the feces. There was an increased calcium elimination as well in the urine as in the feces and also an increased elimination of MgO by both urine and feces. They observe that, while they have been able to compare their data with those of other authors with regard to nitrogen, sulphur, and phosphorus, they have not been able to do the same in the case of the lime and calcium and magnesium And from two cases alone they cannot draw positive conclusions. Repeated experiments through long periods of observation will be necessary to settle the significance of these observations with regard to the calcium and magnesium excretion.

In conclusion they say: "From all that which we have said the relation between the thyroid function and the alterations in the metabolism in Basedow's disease is clear; and the sharp difference between these alterations and those of myxœdema support strongly the thyroid theory of Basedow's disease." The authors also refer to the experiments of Kocher suggested by Scholz with relation to the treatment of this disease by phosphate of sodium, and suggest that in view of their observations it might be well to combine the administration of lime with that of phosphorus.

Auscultatory Ratios in Pulmonary Tuberculosis.—J. S. BILLINGS (*New York Med. Jour.*, 1907, lxxxvi, 10). In the examination of cases of incipient tuberculosis methods of distinguishing between beginning infiltrations of the apex of the right lung and simple physiological intensifications of the breath and voice sounds have been constantly sought for. In cases in which the abnormal signs at the apex seem to be due to a physiological intensification there is a corresponding increase over the lower part of the right upper lobe, both in front and back. In normal chests the same phenomenon at the apex and the lower part of the lobe is noted and a definite ratio of intensification is obtained. The sounds at the apex are of course louder than at the lower part of the lobe, this ratio in health being about 4 to 3 or 5 to 3. In order to record these differences the author used a modification of Oertel's stethoscope (which is illustrated) and examined the patient in a standing posture, listening at the apex of the lung in front and behind and also over the second space and the third rib, one and one-half inches from the sternum in front and just above the upper angle of the scapula behind. Much depends on the manner of speaking and breathing of the patient. Billings' conclusions are as follows: It seems probable that the relative intensity of the breath and voice sounds over the upper and lower portion of the upper right lobe of the lungs remains about the same in health, being about 5 to 4 or 5 to 3; that when the sounds at the apex are physiologically intensified there is a corresponding intensification of the sounds over the rest of the upper lobe, the ratio being the same as in normal chests, namely, 5 to 4 or 5 to 3. In incipient tuberculosis with beginning infiltrations of the right apex the signs here are intensified, while those over the rest of the upper lobe remain normal. The ratio between them is thus distinctly increased to 2 to 1 or even higher. An increase in these ratios to 2 to 1 or over suggests the presence of infiltration, possibly tuberculous, at the apex of the right lung.

Blood Pressure as a Guide in the Treatment of Hemoptysis.—Edward O. Otis (*Boston Med. and Surg. Jour.*, 1907, clvii, 211) considers the varied opinions as to the value of drugs in the treatment of hemoptysis. The ordinary routine treatment of rest, ice, morphine, etc., yields good results in most cases. The efficacy of nitrite of amyl and the nitrites in those cases with a high blood pressure led Otis to test the action of ergot, especially in those cases of low blood pressure or when recurrent and continued slow bleeding might indicate a passive rather than an active hemorrhage. After a large number of blood-pressure determinations by the Janeway instrument the average obtained was found to be about 126 mm. of mercury for tuberculous patients. In those cases upon which the report is based the pressure was between 109 and 119, having fallen from a higher point before the hemorrhage. The estimation of the pressure can be of much use only when routine examinations are made in order that any fall in the blood pressure before hemorrhage may be noted. As a result of his studies Otis concludes that in the treatment of hemoptysis the essential factors are, (1) a thorough knowledge of the blood pressure from previous observations; and (2) the usual routine measures of rest, ice, morphine, and atropine and, depending upon the amount and rapidity of the hemorrhage, inhalations of nitrite of amyl. In those cases of recurrent but persistent bleeding, if the blood pressure be high, use the nitrites, but, if low, ergotin subcutaneously. He says: "I am quite well aware that when I suggest the use of ergot I am uttering heretical doctrine, but in our experience it has apparently proved itself of value in at least a few cases when other remedies have failed."

SURGERY.

UNDER THE CHARGE OF

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Cancer of the Breast and its Operative Permanent Cure.—FINSTERER (*Deut. Ztschr. f. Chir.*, 1907, lxxxix, 143) says that of the cancers of the breast operated on from 1877 to 1903, there was 12.5 per cent. of permanent cures; in the last eight years the percentage increased to 24.64. The extent of the operation was always made to depend upon the local findings. The best method of operation is that which diminishes as well the number of local recurrences, as by the most exact removal of lymph glands it diminishes the occurrence of metastases. An extensive removal of the overlying skin should receive close attention. The supraclavicular fossa should be cleaned out in every case, and absolutely so, when during the operation the infraclavicular glands are found to be diseased or are considered to be suspicious. The question as to whether in the presence of demonstrable disease of the supraclavicular glands, an operation is or is not justified, needs more exact examination. Cases are known which contradict the rule of simultaneous involvement of the supraclavicular and intrathoracic glands and pleura. To decide some cases, one must take into consideration the duration of the disease, the size and situation of the tumor (whether it involves the inner or the outer half of the mamma), the size and possible growth of the supraclavicular glands. On account of the frequency of late recurrences, the time limit of permanent cure should be increased to five years, and for the determination of the results, the ability of the patient to work should be taken into account.

The Actions of the Poisons of Tuberculosis upon the Parenchyma of the Testicle.—MARCOZZI (*Annales des maladies des organes genito-urinaires*, 1907, xxv, 974) says that while the tissues involved by the presence of the tubercle bacilli are the seat of the production of tubercles and necrosis, the poisons of these same bacilli carried in the blood which dilutes them have no necrosing action on the tissues. They do, however, disturb the nutrition and functions of the tissues, producing inflammatory phenomena more or less grave, which are soon followed by different varieties of the process of degeneration. This action of the poisons manifestes itself clinically by a considerable diminution of the vitality of the cellular elements, and it explains the tuberculous cachexia. In the liver it may cause a form of cirrhosis, in the kidney a parenchymatous nephritis with grave changes in the renal epithelium, and lesions in the testicle. Concerning the parenchymatous lesions of the testicles, Marcozzi says that there exists a certain degree of atrophy of the testicle, in consequence of which the albuginea is furrowed. The normal disposition of the cellular elements in the interior of the seminiferous tubules disappears; the testicular cells are intermixed in the wall of the canaliculi. In none of the preparations were spermatazoa or cells of Sertoli met with. The graver cellular changes concerned the spermatic cells, attacked by grave degeneration involving the protoplasm and nuclei. It was observed that even the protoplasm disappeared, leaving only the nuclei, also, changed. The detached protoplasm of the cells accumulated in the middle of the seminiferous tubules. In the nuclei are observed the phenomena which result from alteration in the form of the nuclei, a diminution of the chromic substance, a vacuolization, fragmentation of this substance, which can escape from the nucleus, thanks to the rupture of the membrane, and disappear. In the interior of the tubules are observed masses of protoplasm without nuclei, some polynuclear cells due probably to the division of the nuclei not followed by division of the protoplasm, which not only does not divide, but which progresses to degeneration. In the seminiferous tubules of the testicle and especially of the epididymis, one observes some testicular cells in process of disintegration, in different degrees of degeneration. The wall of the tubule and some of the testicular cells may be detached, and these cells may be eliminated through the tubules at the same time as the spermatic fluid. There are observed, also, some changes in the spermatazoa which, in general, appear in the form of small rods, not having attained the last degree of their development, and presenting some of the residue of the protoplasm of the spermatic cells from which they were derived. There are recognized, also, changes in the heads of certain spermatozoa. Their heads are enormously increased in volume, their tails short, so that they resemble a small nail. Finally, there are some lesions of the intercanalicular connective tissue, which appears atrophied, formed of very rare and delicate fibrils, with small cell infiltration, denoting a certain degree of inflammation, and with large nuclei changed in form or fragmented.

A Case of Polycystic Disease of the Kidneys.—Bartrina and Pascual (*Annales des maladies des organes genito-urinaires*, 1907, xxv, 10) say that the diagnosis of polycystic disease of the kidneys is possible and easy, and with modern methods of renal exploration, we ought always to make it. A polycystic kidney can be the seat of other affections, as lithiasis, tuberculosis, etc. Retention with distention of the pelvis can occur in the absence of any other obstacle to the free passage of urine. In all of the author's cases the renal polycystic disease was associated with a true fibro-epithelial tumor, because the connective tissue and epithelium are developed in equivalent proportions, as shown by the microscopic preparations. The progressive development and indefiniteness of the disease contribute likewise to give to it the characteristics of the tumor. In their cases as in many others, a certain connection can be established between the polycystic kidney of infancy and that of the adult. It seems, therefore, that the congenital origin of the disease is more and more confirmed. As to the etiology of the disease, the authors believe that it should be sought in the embryological development, separately, of one group of embryological epithelial and connective-tissue cells.

Surgical Anatomy of the Hyo-thyro-epiglottic Region.—Poirer and Picque (*Revue de chirurgie*, 1907, xxvii, 1) say that the hyothyroid region deserves attention because it is the path of access to the hyo-thyro-epiglottic region. The bursa of Boyer, which lies between the thyrohyoid ligament and subhyoid muscles, and aids in the movements of the larynx, is far from being constant. It is often represented by a poorly differentiated cellular tissue. The thyrohyoid membrane is made up of a resistant median portion and two thin membranous lateral portions. The epiglottis is connected to the larynx and to the tongue by a true crucial ligament represented in the horizontal plane by the hyo-epiglottic membrane, forming a continuous plane between hyoid and epiglottis; and in the sagittal plane by the median glosso-epiglottic ligament, which below the hyo-epiglottic membrane is attached to the hyo-glossus. The fossa described by Brousses and Brault, as the glosso-thyro-epiglottic fossa, between the thyrohyoid membrane and the epiglottis, is in reality limited to the hyo-epiglottic membrane which closes it constantly and completely above. It, therefore, deserves the name, hyo-thyro-epiglottic fossa. A subhyoid sagittal section always divides it completely into two lateral compartments, each of which represents a triangular prism, the apex being below and the base above. Each one of them contains a fringe of fat, which aids in the free epiglottic movements. It is in these fossæ that the phlegmon of Brousses and Brault develop, the identity of which, however, is yet to be demonstrated. The transverse incision of Malgaigne, large and early, is considered to be the treatment for it. In case it progresses toward the base of the tongue, the transverse incision of Vallas, will permit a wide exposure for view.

Volvulus of the Small Intestine and of the Initial Portion of the Large Intestine.—Guibe (*Revue de chirurgie*, 1907, xxvii. 91) says that cases of volvulus of the small intestine ought to be operated on soon. Leichenstern has claimed that the intestinal twist can undo itself; but those who have seen these cases know that nothing is less likely. They are always very tightly locked. To attempt to bring about an untwisting would lead to a fatal termination by a failure to undo the twist or by rupturing the intestine. Laparotomy should be done and it should be sufficiently large to explore conveniently the abdomen and the lesions, and to permit one to undo the volvulus. Recognition of the lesion is easy enough when the volvulus only involves a small loop of intestine; when it is a little long and the intestine above is distended, it can be very difficult of recognition. When recognized it should be emptied by puncture or enterotomy. With the emptying of the involved intestine, the untwisting results at the same time. If the condition of the intestine calls for resection the prognosis is very bad. Sometimes the volvulus begins with the signs of a strangulated hernia and then the diagnosis is not easy. In such cases the operation is usually begun as a herniotomy. If the volvulus and strangulation are in the hernial sac the diagnosis is easy as soon as the sac is opened. The diagnosis is not so easy when the sac contains only a part of the twisted intestine. On opening the sac the intestine may be found of a reddish-black color or even gangrenous. The hernial ring is seen not to be constricting the intestine and the cause must be sought higher up in the abdomen. If there is, also, a true strangulation at the neck of the sac this can be relieved in the ordinary way, but if on examination of the intestine it is seen that the strangulation extends higher, the intestine is drawn out until the seat of the trouble is found, extending the original herniotomy incision. An additional median incision is to be preferred. The herniotomy wound is to be closed or not according to whether the condition of the contained intestine will permit it or not.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Serum Therapy of Bacillary Dysentery.—VAILLARD and DOPTER (*Revue de thérap.*, 1907, x, 331) consider that antidysenteric serum injected in doses adapted to the patient in hand will relieve the intestinal disturbances of mucous colitis almost immediately and ensure a rapid recovery. The action of the serum is most prompt and effective when it is administered early in the disease. It is also effectual late in the affection, arresting the progress of the infection and hastening cure. In fine, antidystenteric serum is the specific agent in the treatment of bacillary dystentery, and in this disease has an effect analogous to that of antitoxin in diphtheria; its universal employment will reduce the mortality from dysentery to a minimum. By its administration the symptoms become rapidly ameliorated and a prompt cure is brought about. As a prophylactic its use is important and, especially in epidemics, it should be given as a routine. The above conclusions are the result of an extensive study of patients treated by the serum. The dosage in mild infections is from 5 to 7½ drams, but in grave instances of the affection it may be necessary to inject as much as 2½ ounces and to repeat the dose on the day following. As the treatment shows its good effect and the defecations become fewer, the injections may be continued, but the dosage should be gradually diminished.

The Hydrotherapeutics of Mucous Colitis.—WILSON (*Edin. Med. Jour.*, 1907, xxii, 15) states that the use of balneotherapeutic measures for the treatment of mucous colitis is attended with excellent results, often after the usual methods have met with little or no success. The baths consist of two parts, which may be given together or separately as the physician may direct: (1) The intestinal douche; this is administered while the patient lies on a special couch. A rectal tube is passed high into the bowel and the prescribed amount of hot mineral water is allowed to flow, under carefully regulated pressure, into the intestine. While the water is retained the patient's position is changed; the bowel is later emptied and the process is repeated. (2) The Tivoli douche; this consists of an ordinary reclining bath, in which the patient lies covered, except for the head and neck, in warm mineral water. While so lying a hot-wave douche is played on the abdomen; this latter is usually of a much higher temperature than that of the bath. These methods are carried out in conjunction with special dieting and the drinking of mineral waters. The technique described is that employed at the springs of Harrowgate, England.

The Treatment of Itching.—BULKLEY (*Jour. Amer. Med. Assoc.*, 1907, xlix, 321) divides the useful external applications into two types, the soothing and the analgesic. Of the former he suggests the following formulæ: ℞.—Phenol, ʒss–ʒj; pulv. calaminæ prep., ʒj; zinci oxidi, ʒij; glycerin, ʒiij; aq. calcis, ℥j; aq. rosæ, q.s. ad ℥iv. Sig.—To be sopped on repeatedly. ℞.—Picis liquidæ, ʒiv; potassii causticæ, ʒij; aquæ, ℥x. Sig.—Dilute one to ten or twenty with water and bathe the surface, applying a suitable ointment afterward. Ichthyol in ointments or in 10 to 25 per cent. solution in water or oil is often useful. For mild general pruritus the following ointment may be prescribed: Phenol, 20 to 40 grains; lanolin, 1 ounce; boroglycerin, 4 drams; ung. aq. rosæ, 3 ounces. One-half to one dram each of camphor and hydrated chloral rubbed up with lanolin to an ounce is an efficient antipruritic, but it is irritating to the broken skin. Many skins do better without greasy applications, and the glycerite of starch may be substituted in the above ointments for the fatty base. Tar, oil of cade, and oil of birch are also valuable and harmless antipruritics.

The Treatment of Delirium Tremens.—EICHELBERG (*Münch. med. Woch.*, 1907, xx, 978) believes in the immediate suppression of alcohol in delirium tremens and in the employment of hydrotherapeutic measures rather than of hypnotics; the former serve to increase and to maintain the activity of the heart, although one would expect an opposite effect. In instances of cardiac weakness stimulants, strophanthus, digitalis, camphor, caffeine, are employed, and in about three days, when the delirium begins to lessen, 30 to 60 grains of chloralformamide are given; this quickly induces sleep. Thirst is controlled by bitter infusions. If pneumonia appears as a complication, digitalis and alcohol are administered. In these patients the prognosis is distinctly bad.

The Treatment of Diphtheritic Paralysis.—COMBY (*Bulletin méd.*, 1907, xxxviii, 442) considers that every patient, old or young, affected with diphtheritic paralysis, light or severe, recent or of long standing, localized or general, should immediately receive injections of antidiphtheritic serum. These should be repeated daily for from three to six days, depending on the severity of the affection. The dose employed should be from 150 to 300 minims of Roux's serum. Such dosage is followed by no unpleasant effects and should be administered to all patients, even those who received antitoxin during the acuity of the disease. He employed this treatment in thirteen patients, all of whom were cured.

Opsonic Therapy in Skin Diseases.—Varney (*Jour. Amer. Med. Assoc.*, 1907, xlix, 316) concludes a report upon a series of patients affected with acne, furunculosis, sycosis, and whom he has subjected to vaccine treatment, with the following statements: In bacterial inoculations we possess a therapeutic agent with a specificity of great merit. Wright's method of obtaining the resisting power of a patient is also of unquestionable value as a guide to determine what will raise the resistance. Vaccines made from the patient's own invading microorganisms produce better results than stock vaccines. These results may be obtained with or without opsonic readings, but they will be better if controlled by frequent estimation of the patient's resistance. Proper vaccination, administered in appropriate doses and when the opsonic index indicates the necessity, is an efficient treatment of certain chronic local infections.

Vaccines in Surgical Therapy.—Bergey (*Univ. Penn. Med. Bull.*, 1907, xx, 50) states that bacterial vaccines may be employed as protective agents in surgery when it is evident that proper asepsis is impossible, and in patients of lowered vitality when the probability of secondary pyogenic infection is evident. In operations about the mouth, for instance, when it is impossible to control secondary infection, it is advisable to subject the patient to pre-operative inoculations so as to increase his opsonic index for staphylococci and streptococci. In diabetics and albuminurics, when the general vitality is low, it is believed that the success of surgical procedures can be increased by first raising the subject's opsonic index for the common pyogenic bacteria. Similarly secondary infection of tuberculous abscesses may be avoided. The use of the vaccines seems clearly indicated in accident wounds when asepsis is doubtful and when mutilation of tissues renders secondary infection probable.

Nuclein in the Prophylaxis and Treatment of Peritoneal Infections.—Chantemesse and Kahn (*Klin.-ther. Woch.*, 1907, xiv, 663) advise the employment of hypodermatic injections of the sodium salt of nucleinic acid in the prevention and treatment of peritonitis, on the ground that this substance has the property of increasing the number of leukocytes in the circulating blood, and thus increases the bodily powers of resistance to infection. Experimentation in man as well as in animals shows that after the injection of this substance a mononuclear leukocytosis takes place which reaches its height in about forty-eight hours and lasts from four to five days. An injection of 6 grains of sodium nucleinate in about 10 drams of sterile salt solution into the flank or into the fleshy part of the thigh may be painful and is likely to be followed in twenty-four hours by the signs of inflammation; these soon disappear. The injection may be repeated two or three days later and as many as three injections may be given in a single day. The leukocytes may thus be increased to twice or three times their normal number. In one patient with intestinal perforation who was treated by the nuclein injections, the opsonic index was increased from 1.6 to 2.5 twenty-four hours after these had been undertaken. Chantemesse and Kahn cite patients suffering from enteric fever in whom all the signs of intestinal perforation were present and in whom all these manifestations disappeared after the nuclein injections. It is possible that in these patients a small intestinal perforation may have been present, but it is difficult to be sure of this. The treatment is recommended in enteric fever with symptoms of perforation or peritonitis; it is suggested that it may give benefit in such conditions as pneumonia and erysipelas, and that it may be employed in the prophylaxis of operative peritonitis.

Formic Acid in Diphtheria.—Ker and Croom (*Edin. Med. Jour.*, 1907, vi, 487) have employed formic acid in 412 patients during 1906 at the Edinburgh City Hospital, with the result of diminishing the death rate by 1.8 per cent. over that of the previous year. Previously strychnine had been given as a heart tonic, but during 1906 formic acid in 25 per cent. aqueous solution, in doses of 5 to 20 minims every four hours, was substituted, the dosage being graduated rather by the severity of the infection than by the age of the patient. No change in the heart action was noted until after about forty-eight hours, and then the change was rather a negative one, that is to say, many of the severe infections did not show the expected cardiac weakness and irregularity; on the other hand, the pulse in many instances was much improved in character, as was the color of the skin and the general nutrition. Patients were observed who appeared doomed to die of heart-failure, but who rallied, this manifestation being attributable to the possible limiting effect of the formic acid upon the degeneration of the heart muscle or its beneficial action upon the undamaged muscular tissue. The most striking result of the treatment was the diminution of the number of instances of paralysis, the percentage being only 2.9, as against 9.09 in the previous year, which was the lowest in several years. If this diminution of the paralysis is really due to the formic acid, the point is raised why a muscular tonic should have such a result on a lesion usually regarded as primarily of nervous origin, although it is true that post-diphtheritic muscular weakness is rather a paresis than a complete paralysis. They conclude that the results obtained are distinctly encouraging, particularly in relation to the occurrence of cardiac failure and paralysis. Formic acid is, at least, an admirable tonic; it is safe and may be employed to the advantage of the patient.

PEDIATRICS.

UNDER THE CHARGE OF
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Scarlatina and Duke's Disease.—A. C. Cotton (*Jour. Amer. Med. Assoc.*, 1907, xlix, 1417) believes in the entity of the fourth, or Duke's disease. He summarizes its clinical features as follows: The disease is less contagious than measles and probably equal to that of scarlatina; its period of transmissibility is from two to three weeks. The period of incubation is from one to three weeks. Prodromas are often wanting, the first symptom being frequently the rash itself. Febrile symptoms may precede the rash by from six to twenty-four hours. Initial vomiting is exceptional. Mild catarrhal symptoms with hyperemia of the mucous membranes of throat, mouth, or eyes are occasionally present. The eruption appears first on the face, spreads to the neck, and then rapidly downward, involving the trunk and portions of the extremities in a few hours. It is especially pronounced over areas covered by clothing and does not pick out the flexor surfaces. Fine points may first appear, quickly blending into a general hyperemia; small patches of normal skin with irregular margins are visible here and there. The oronasal pallor of scarlatina is absent. The rash fades rapidly after two to three days without stain, which is often observed in rubella and measles. There is no itching and the skin does not feel hot. Desquamation of a fine and branny character usually follows the disappearance of the exanthem. The fever subsides with the eruption. The tongue is negative beyond an occasional coating, the throat, beyond an occasinal hyperemia; the pulse is in proportion to the fever. The postcervical and occipital lymph glands may be enlarged in the early part of the attack; massive enlargements are only seen in complicated cases. Sequels and complications rarely occur, the course being usually mild and uneventful. It is differentiated from scarlet fever by (1) its long period of incubation; (2) absence of initial vomiting; (3) moderate fever of brief duration; (4) normal ratio of pulse to temperature; (5) absence of characteristic scarlatinal tongue; (6) absence of, or, if present, the fine character of, the desquamation; (7) the freedom of sequels; (8) probably the absence of leukocytosis.

The Prevention of Ophthalmia of Infancy.—L. Howe (*Virginia Medical Semi-Monthly*, 1907, xii, 265) points out that more blindness is caused by ophthalmia neonatorum than by any other disease and that a 2 per cent. solution of silver nitrate is the most reliable prophylactic. He emphasizes the necessity of a law in all States which makes necessary an immediate report of all infants in whom one or both eyes become inflamed, swollen, or reddened at any time within two weeks after their birth; and if laws were passed to make the employment of Credé's method compulsory, it would be a still greater boon to the human race.

The Purpura of Children.—H. R. Dean (*Brit. Med. Jour.*, 1907, ii, 815) has studied 52 cases of primary purpura during the last ten years and has found them all to be of one type with differences in degree of severity. Some, because of their mildness, had to be classed as purpura simplex; but even they presented some intestinal derangement and joint disturbance. In some of the cases a predominance of arthritic changes was noted, but in none of these could a history of rheumatic fever be obtained. The joints are not so hot as in rheumatism; a predominant feature is a general œdema of the shin and foot, while the joint disturbance is not so obvious; the temperature chart of the two diseases differs; profuse sweats are absent in purpura and cardiac complications are almost unknown. Gastro-intestinal symptoms, such as sudden violent pain, vomiting, bloody diarrhœa, are extremely common, and may precede the purpuric rash for some days or even weeks. In purpura the lymphocytes are often increased; otherwise the blood picture is that of sudden and severe hemorrhage. Hematuria was noted seven times, and albuminuria without hematuria twice; hematuria may persist for weeks. In some instances the renal symptoms are sufficiently severe to permit of a diagnosis of acute renal disturbance. In 1 case the symptoms of renal calculus were simulated. Hematemesis occurs only occasionally; in 2 cases the symptoms pointed to intracranial hemorrhage, possibly meningeal; in 1 there was effusion of blood into the pleural cavity. Hemorrhage into the buccal mucous membrane and conjuntiva, as well as epistaxis, are seen occasionally; retinal hemorrhage is rare. One-third of the patients have enlargement of the liver; the spleen is but rarely enlarged. Dean believes purpura to be the result of some undiscovered infective or toxic process. A very characteristic condition is to be found in the recurrence of the purpuric symptoms at irregular intervals for several weeks or even months. Through 4 cases of the series the close relationship of purpura with hemophilia is pointed out; but in all 4 the history of hemorrhage was on the father's and not, as in hemophilia, on the mother's side.

Case of Cerebral Hernia in a Boy.—E. F. TALBOTT (*Jour. Amer. Med. Assoc.*, 1907, xlix, 1441) reports the case of a boy, aged sixteen years, who as a result of a gunshot wound in the head had a paralysis of the right arm and leg, and of the bladder and rectum. There were two openings to the left of the crown of the head about one and one-half inches apart. A depressed portion of the inner table was removed, together with several spicules of bone, and the wound was closed with drainage. Pressure symptoms necessitated reopening of the wound; a hernia resulted, but the pressure symptoms were relieved. A sinus leading into the brain discharged pus for a month, when a small piece of bullet was found in its bottom; its removal caused diminution in the discharge of pus, but an increase in the size of the hernia. The hernia had to be cut away because of pressure symptoms, but later reappeared larger than before. Five months after the injury an abscess was located and drained; later still the hernia had again to be removed, when a second abscess which led well into the substance of the brain was opened and drained. Since then the wound has entirely healed, the boy enjoying good health physically and being well mentally. The bladder and bowels have recovered, but the forearm and leg are still paralyzed.

Retropharyngeal Abscess.—H. I. PINCHES (*Brit. Med. Jour.*, 1907, ii, 813) includes collections of pus at the back and side of the pharynx under this heading. He differentiates between the acute and chronic, the non-tuberculous and tuberculous. Most of the acute abscesses occur during the first twelve months of life; the children as a rule have been perfectly well and strong. In most cases the condition is preceded by some inflammatory affection of the tonsil or some of the lymphoid tissue of the neighborhood; enlarged tonsils and adenoids are present in almost every case of retropharyngeal abscess. The symptoms are: difficulty in swallowing and breathing; the child is always very ill, having high fever, a rigid neck, a retracted head, a peculiar cry, and in most cases a nasal discharge. Examination of the throat nearly always makes the diagnosis apparent, and particularly if digital examination is used. The finger should be used very gently, however, for fear of rupturing the abscess. If diagnosticated early the prognosis is usually good; if undiagnosticated the abscess ultimately discharges into the throat and septic bronchopneumonia terminates the case. A large incision through the pharyngeal wall is preferable to other routes of operation. The author closes by stating that the condition is very frequently overlooked, possibly more often than any acute condition of childhood, owing to the variability and multiplicity of the signs and symptoms accompanying it. If the pharynx is examined the diagnosis is made in every case.

Prolapse of the Rectum in Children.—P. L. Mummery (*Brit. Med. Jour.*, 1907, ii, 812) states that rectal prolapse is a comparatively common affection among children. Of 50 consecutive cases he has found 22 to be among males, 28 among females; their ages varied from three months to five years. Of causes diarrhœa was the commonest precursor of prolapse, occurring in 14 of the cases; 13 of the patients had adenoids; 3 had worms; in 3 the condition followed whooping-cough and in 3, measles; 2 had rectal polyps, 1 prostatic inflammation, and 1 a rectal stricture; in 1 case it was blamed on constipation; stone in the bladder or phymosis was not present in any of the cases. General weakness and malnutrition, therefore, caused the larger number, local causes being present in but 7. Absorption of fat is a result of malnutrition, and the removal of this fat from the rectum predisposes to prolapse. The exciting cause he considers to be the unnatural method of defecation adopted in civilized countries. Pathological prolapse is unknown among animals and uncivilized races. The natural position for defecation is the squatting one, in which the glutei and perineal muscles are firmly contracted, thus supporting the levator ani and tightening the pelvic fascia; the coccyx is firmly fixed in the squatting position, the lower part of the rectum thus forming a considerable angle, while in the sitting posture the rectum is almost a straight tube. The obliquity of the false pelvis in children also predisposes to prolapse. In treatment the child's general health and nutrition must be improved. The stools should be passed in the squatting position into a shallow pan. This cures prolapse as a rule at once. Local causes, whether rectal or otherwise, must be attended to.

OBSTETRICS.

UNDER THE CHARGE OF

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Valvular Disease of the Heart Complicating Pregnancy.—Newell (*Surg., Gyn., and Obstet.*, May, 1907) contributes a paper upon valvular diseases of the heart complicating pregnancy, reporting 3 cases.

The first was a multipara six and a half months advanced in the sixth pregnancy. Compensation began to fail ten months before admission to the hospital. Upon the advent of pregnancy the symptoms grew progressively worse. On admission the patient was anemic, with labored breathing, moderate œdema of the lungs, œdema of the extremities and abdominal wall. The heart was much enlarged, with diffuse pulsation over the apex; the heart rate 140, pulse 112. A loud, systolic murmur was heard over the entire chest. The uterus extended three fingers' breadth above the umbilicus; the child was evidently not large. The abdominal cavity contained some free fluid. The patient's condition was so bad that immediate operation seemed excessively dangerous. Accordingly, delay was practised to secure a better condition. As the delay resulted in but very slight and transient improvement, the patient was given ether, the uterus dilated, and the child delivered by version. Death occurred before the completion of the operation, which occupied but sixteen minutes. A small, living child, weighing two and a half pounds was born, but died in a few hours. During the six and a half months of her pregnancy this patient had been under the care of eight different physicians, who had attempted to stimulate the heart and secure compensation, not realizing that the added strain of pregnancy made radical improvement impossible. Had pregnancy been interrupted at an early stage the patient's life might have been indefinitely prolonged.

The second case was that of a primipara, with enlarged heart, slight cyanosis, marked dyspnœa, and slight pulmonary œdema. This patient had a presystolic murmur, with marked thrill over the apex. With rest, digitalis, and strychnine, the patient improved, compensation being reëstablished, and the patient passed comfortably through the remainder of her pregnancy to full term. The patient passed through her first stage of labor with comparative comfort, and the perineum softened to some extent. Collapse, however, developed, the patient became very cyanotic, œdema of the lungs developed, and the pulse rose to 160. The child was immediately delivered by forceps, and the patient was greatly relieved by moderate bleeding. She made a fair recovery. Although the patient apparently did well in labor, her final collapse showed that her favorable progress was apparent only, and that interference should have been undertaken earlier.

The third patient was a primipara, aged thirty-five years, with a history of rheumatism, endocarditis, and aortic insufficiency. She passed through pregnancy fairly well, and was seen in consultation after forty-eight hours of moderate labor. Morphine had been given to lessen the patient's suffering. The patient was well developed and well nourished. The heart was greatly enlarged, with a loud systolic murmur over the aortic region. The radial pulse was poor. The uterus was rigidly contracted about the child, with a contraction ring palpable at the child's neck. The fœtal heart was 160. On vaginal examination the lower segment was greatly thinned; the os was dilated to about one-half inch with a wire edge. The cervix was tightly applied over the child's head. Engagement was present. Under ether the cervix was dilated manually, the occiput rotated in front, and the child extracted by forceps. The child was living at birth, but died in five or six hours. The mother was in a fairly good condition at the end of the operation and reacted, but collapsed and died in four or five hours without hemorrhage. Here again, while the patient's compensation carried her through pregnancy, forty-eight hours of labor was more than the heart could endure.

Newell concludes that an organic heart lesion in a pregnant woman calls for constant watchfulness, and should compensation begin to fail at any time in pregnancy an attempt should be made to restore compensation by rest and tonics, but unless these measures are promptly successful, pregnancy must be interrupted. In all such cases labor must be made as brief as possible, and terminate promptly in the interest of the mother.

Tubal Gestation with Continued Growth of the Fœtus after an Hematocele had Developed.—Fairbairn (*Jour. Obstet. and Gyn. Brit. Empire*, December, 1906) reports a very interesting case in which a patient had typical signs of ruptured ectopic gestation. Her pain grew gradually better and hemorrhage ceased, but returned profusely. On examination, the uterus was slightly distended and so rigid and tender that it was very difficult to make out the position of the womb. Upon operation the omentum and underlying viscera were matted together by adhesions, suggesting a condition of inflammation. There was yellow lymph among the adhesions, and a dirty, almost purulent fluid escaped from the abdomen. After separating the adhesions the swelling was found to be the left Fallopian tube with an hematocele sac. During the removal of the mass the sac ruptured, followed by the discharge of the fœtus, placental tissue, and blood clot. The cavity in the pelvis was drained by gauze, and the patient made a good recovery. Upon examination of the specimen, abundant evidence was found that the embryo had continued to develop after rupture of the old sac, with formation of a pelvic hematocele.

Acute Salpingitis Complicating Tubal Gestation.—Eden (*Jour. Obstet. and Gyn. Brit. Empire*, December, 1906) reports the interesting case of a woman with tubal gestation, complicated with acute salpingitis. Upon opening the abdomen, omentum and coils of small intestine were found adherent to a pelvic tumor. This was gradually isolated and its pedicle traced to the right broad ligament. The pedicle was clamped, and the tumor removed. There were many adhesions, recent and very vascular, and oozing from the bed of the tumor was arrested by packing. The left uterine appendages were found to be practically normal. The patient made a good recovery. Upon examining the specimen, the gravid Fallopian tube was also the site of an acute salpingitis. It is difficult to understand how the ovum can implant itself upon the mucous membrane of a tube in a state of acute inflammation. The most rational explanation is found in the fact that conception and infection must have occurred at about the same time.

Ruptured Ectopic Gestation Following the Alexander Operation.—Batchelor, (*Jour. Obstet. and Gyn. Brit. Empire*, December, 1906.) reports 4 cases of ruptured ectopic gestation occurring in patients upon whom had been performed the Alexander operation. It seems difficult to trace the cause of relationship between the ectopic gestation and the Alexander operation.

Tubular Mole Bemoved During the Process of Operation—DORAN (*Jour. Obstet. and Gyn. Brit. Empire*, December, 1906) reports the case of a patient who came to the hospital complaining of abdominal pain. There had been a slight discharge of blood almost daily. Some time before coming to the hospital she had an attack of violent pain in the lumbar region, followed by painful micturition. Upon operation, the left appendages were bound down by adhesions, but were apparently healthy, the Fallopian tube being unobstructed. The adhesions were freed and the tube allowed to remain. The mass in the pelvis proved to be the distended right Fallopian tube, which was removed with the ovary. Upon examining the specimen, it was found that tubal abortion was taking place.

Twin Pregnancy in a Fallopian Tube.—MCCANN (*Jour. Obstet. and Gyn. Brit. Empire*, December, 1906) observed a tubal twin pregnancy. When seen the patient was exceedingly pale, unable to reply to questions, and with a very rapid and weak pulse. The lower abdomen was distended and sensitive, and a tumor was detected in the left iliac region. The uterus was slightly enlarged. The abdomen was opened, with a diagnosis of tubal pregnancy. The dilated left Fallopian tube and a mass of blood clot about it were removed. On examining the specimen in the dilated tube there was found a cavity occupied by two fœtuses. They had separate umbilical cords and were unequal in size.

GYNECOLOGY.

UNDER THE CHARGE OF

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Congenital Atresia of Both Fallopian Tubes.—This is an extremely rare condition if one is to judge from the sparseness of the subject in literature and from a fairly large clinical experience. A case somewhat doubtful is reported by DOLERIS (*Ann. de gynec. et d'obstet.*, August, 1907, p. 465). The subject was twenty-eight years of age, large, well developed, of full habit, and in excellent health. Menses began at the age of fourteen years, was of an irregular type, painless, and without marked periods of real amenorrhœa. It lasted four days and was of a normal amount. She was married at the age of twenty-five years, when her menstruation became perfectly normal. She was sterile during this marriage. At the age of twenty-six years she was again married and now had some pelvic pain, at times severe. Eighteen months later she consulted Doleris for treatment of sterility. He found she had a normal-sized uterus that was retroverted, and appendages slightly enlarged and prolapsed. The tubes were non-adherent, as was the uterus. The muscular coats of the left tube were found to be thickened and hard near the uterus, but at 3.5 cm. from the uterus the canal was constricted abruptly with very thin and soft walls. It was impervious; distal to this the tube was enlarged to the size of a walnut and contained sebaceous matter.

For a distance of 3 cm., beginning 2.5 cm. from the uterus, the right tube was much thinned and impervious; in fact, macroscopically no trace of the tube could here be found. Beyond this portion it seemed normal. A small fibroid was found in the uterine wall in the cornu just behind the tubal insertion. The possibility, even the probability, of a former salpingitis is to be suspected, both from the findings and the severe pain the patient had experienced. That a developmental abnormality antedated and had a causative effect upon the inflammation cannot be successfully disputed.

These possibilities are also illustrated in a case of congenital atresia of the left tube reported by Lejars (*ibid.*). The right appendage of a girl, aged seventeen years, was a pus tube and an ovarian cyst, both of which were adherent to the omentum and to the uterine fundus. The left tube, ovary, and border of the uterus from the cervix to the cornu had the appearance of a bridle or band of tissue attached to the cervix at one end and at the other to the wall of the pelvis, well separated from the fundus. The structure was fibrous and ended in a small pavilion near which was a very small microcystic ovary. Microscopic study of the tube showed it to be impermeable except for a very short distance at the fimbriated end. This tube was clearly one the development of which had been arested early in fœtal life, the cord-like body evidently being the part of the left Müllerian duct that forms the Fallopian tube and left half of the uterine body.

The Diagnosis and Treatment of Genito-urinary Tuberculosis with Koch's Tuberculin.—R. Brinbaum (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1174) states that he has used Koch's tuberculin for diagnostic purposes in 100 cases, with satisfactory results, as judged by the subsequent history or operative procedure; 23 patients have been treated with either the new or the old tuberculin of Koch for such conditions as tuberculosis of the appendages and tuberculosis of the bladder with marked improvement or cures in many instances.

Migration into the Bladder of an Artery-clamp Left in the Abdominal Cavity. W. Stoeckel (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1105) says a laparotomy was performed upon a woman in October, 1906, and a hemostat evidently left in the abdomen. About eight months afterward the patient complained of pain upon urination, and vaginal and cystoscopic examinations revealed the presence of an instrument partly within the bladder. A colpocystotomy was performed and the instrument, which was found to lie in part in a walled-off abscess posterior to the bladder, was removed.

Perforation of the Uterus.—R. v. Braun-Fernwald (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1161) reviews the reported cases of perforation of the normal and the pathological uterus by the use of sounds, curettes, bougies, irrigators, forceps, and other instruments, and discusses the diagnosis and treatment of perforation. Mention is made of a case coming under his own observation in which the end of a glass catheter believed to have been used for producing criminal abortion was removed from an abscess in the abdominal cavity in the region of the sacrum.

Menstruation without Ovaries.—G. GELLHORN (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1195) reports two cases. In the first the ovaries were removed in two operations separated by an interval of one year. Menstrual periods were regular and normal after the first operation. For several months after the second oöphorectomy they were of short duration, but gradually lengthened to three days and were, in all respects, normal. A third laparotomy was performed by Gellhorn, himself, seventeen months after the second operation. No trace of ovaries or tubes was found. The uterus was somewhat atrophied. Three bands connected the fundus of the uterus with the omentum. These adhesions were cut and menstruation cased.

The second case was that of a woman, aged thirty-five years, who, six years previous to consulting Gellhorn had had the left adnexa removed. Her menstrual periods had been regular since her seventeenth year and of about five days' duration. Gellhorn's diagnosis—retroversion of the uterus, tumor of the right tube, perimetritis, and parametritis—was confirmed upon operation, at which the uterus was freed from all adhesions, the right adnexa removed, a ventral suspension of the uterus performed, and the appendix removed. The patient made a good recovery from the operation and was free from pain. Climacteric symptoms soon appeared, which were not satisfactorally controlled by bromide and other drugs. Gellhorn, therefore, administered ovarian substance in the form of 0.3 gram tablets three times daily, beginning January 17. On March 5 the patient had a bloody flow from the uterus, the source of which was ascertained by a speculum examination and which continued for five days. A second flow of the same character occurred on March 22. The ovarian tablets were then discontinued until April 17. Two days after resuming the treatment the third flow commenced. A fourth began May 1 and a fifth June 1. Treatment was then discontinued for six weeks and the flow did not appear during that time. Five days after resuming treatment a second time the flow reappeared. The most frequent cause of menstruation and even troublesome and irregular menstruation after ablation of the appendages is an infected endometrium or uterus, though many other causes are known. In Gellhorn's first case adhesions to the omentum were clearly proved to be the cause. His suggestion of extra blood supply is probably correct. In his second case ovarian substance seemed to have a causal relation that has not been referred to before.

Blastomycetes and Inflammations of the Female Genitalia.—VAN DE VELDE (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1135) has made a study of the flora of the normal and diseased female genitalia and has in preparation a detailed account of his work. In this article attention is called briefly to some of the principal results obtained. In 77 instances the author has isolated blastomycetes from inflamed tissue, and in 24 of these cases the organisms have been grown in various artificial media. In the remainder of the 77 cases the yeasts have died out after a few generations, or their growth has been limited to special media. Of the 24 species carefully studied 13 have not hitherto been described.

None has been found in normal genital organs. The greater number have been isolated from cases of acute cervicitis with vaginitis and vulvitis. A few have been obtained from cases of chronic cervicitis. In a typical case of beginning mastitis the author found in the milk of the affected breast no organisms other than blastomycetes and these apparently belonging to one species. The infant nursed by this patient had a diarrhœa and in the stools were found the yeast cells. Several months later the same patient had an inflammation of the genital organs and in the secretions yeast cells were present in large numbers. Chronic gonorrhœa and pregnancy are believed to offer favorable conditions for blastomycetic infection. In the blood of a woman dying of puerperal sepsis and also in the lochia, Van de Velde found a large number of yeast cells in addition to bacteria. Other cases of a similar nature are mentioned. The results of the investigations are considered of sufficient importance to warrant further investigations, not only in regard to chronic endometritis, but other inflammatory conditions.

Statistics of Prolapse Operations.—J. Scharpenack (*Zentralbl. f. Gynäk.*, 1907, xxxi, 1073) discusses the results secured in 100 operations for prolapse by the Wertheim-Schauta method, in which a longitudinal incision is made in the anterior vaginal wall, the peritoneum separated from the cervix and bladder, the fundus pulled down through an opening in the uterovesical pouch and sutured to the edges of the vaginal incision, and the posterior surface of the uterus sewed to the posterior wall of the bladder. A plastic operation is also performed on the perineum. Of the 45 cases examined by Scharpenack eight months to three years and five months after the operation, perfect objective results were found to have been secured in 21 ca es. A slight anterior prolapse was found upon strong bearing down in 16 cases and a slight posterior prolapse in 3. The results were unsatisfactory in 5 cases. Except in 2 cases, however, the patients were entirely satisfied. In 57 cases out of 72 favorable reports were made by the patients as regards subjective symptoms. In 4 cases improvement was admitted, but in 11 the symptoms were no better after the operation. Many of the reports were made by correspondence and not very much importance is attached to them. Those personally examined were, on the whole, extremely satisfactory. The operation is highly commended in preference to pessaries, especially when the tubes are cut, as was done in every instance by Scharpenack, and when the woman had passed the climacteric.

DERMATOLOGY.

UNDER THE CHARGE OF
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The Relationship Between Diseases of the Skin and of the Kidneys.—GLASERFELD (*Dermatologische Zeitschrift, Band* xii, *Heft* 10) after a careful study of the literature of diseases of the skin and kidneys as to their association with one another, concludes that only in inflammations and disturbances of the circulation of the skin, do affections of the kidneys occur which are dependent upon the former. Such are almost always found in impetigo herpetiformis, and often in erythema, urticaria, acute circumscribed œdema, Raynaud's disease, purpura, eczema, and acute pemphigus. The kidney symptoms appear during the existence of the skin affection, or immediately after it. They are usually of a harmless character; but in eczema and purpura the prognosis of the nephritis may become more serious. Progressive and regressive disturbances of the nutrition of the skin, infectious diseases, and parasitic dermatoses were never observed to be produced by nephritis; with the exception of pruritus, it was only disturbances of the circulation and inflammations of the skin which were noted. Acute nephritis predisposes very little to complications on the part of the skin. The several forms of chronic nephritis vary little as to their liability to be followed by affections of the skin. The latter appear only after disease of the kidneys has lasted for some time, and are very difficult to cure, improvement only appearing with the adoption of a therapy suited to the kidney affection.

The Histology and Roentgen Therapy of Mycosis Fungoides.—HERXHEIMER and HUBNER (*Archiv f. Dermat. und Syphilis,* Band lxxxiv, Heft 1–3) report a series of 10 cases of mycosis fungoides with a study of the histology of 1 of them, both before and after treatment with the Röntgen rays. They were able to establish the fact that the mycosis infiltrate gradually entered the skin from the lower layers of the corium, and could distinguish the small mononuclear mycosis cells as specific structures. The vessels were greatly altered by endarteritis, and the connective tissue had undergone a peculiar (hyaline?) degeneration. The sweat glands were walled about and filled up with the tumor mass, and their epithelium altered and partly destroyed. After a short radiation there was exfoliation of the epidermis and infiltration of the whole tissue with polynuclear leukocytes, leading to a partial disappearance of the mycosis cells and of the peculiar changes in the connective tissue. After a longer radiation there was beginning regeneration of the epidermis with a great number of new-formed bloodvessels, and the tumor cells were reduced to a scanty remnant. The sweat glands were normal, but the connective tissue exhibited a peculiar degeneration.

The authors believe the histological changes which they have found prove that the Röntgen rays are able to destroy the specific cells of myocsis fungoides, and are, therefore, to be regarded as a specific curative agent in this disease. They believe that we now possess a method of treatment which, employed at the right time, often acts curatively, and makes possible a considerably more favorable prognosis.

Association of Erythematous Lupus and Tuberculous Lupus.—Spitzer (*Annales de dermat. et de syphiligraphie,* 1907, No. 3) reports the following case observed in Lang's clinic in the Vienna General Hospital: A man, aged thirty-seven years, was admitted to the hospital in January, 1900, having upon both cheeks plaques with a cictricial appearance and slightly red and infiltrated border, and in places covered with very adherent scales. Here and there in the midst of the cicatrix were isolated or confluent nodules, undoubtedly the tubercles of lupus vulgaris. Four years before the patient had been admitted to Kaposi's clinic with a diagnosis of erythematous lupus, and had been treated with more or less success by applications of "gray plaster," salicylated plaster, curettement, and injections of arsenious acid. In addition to the lesions upon the skin the buccal mucous membrane presented grayish streaks extending from the labial commissures toward the pharynx, which were diagnosticated erythematous lupus. Microscopic examination of various parts of the disease confirmed the diagnosis of erythematous lupus associated with tuberculous lupus.

A Case of Xeroderma Pigmentosum Without Pigmentation.—Audry (*Annales de dermat. et de syphiligraphie,* 1907, No. 3) has observed a case of xeroderma pigmentosum which he reports in detail, in which there was practically absence of pigmentation, the only pigment anomaly being a few pale brownish and yellowish, freckle-like spots upon the backs of the hands. The patient was a man, twenty-four years old, who entered the hospital on account of an epithelioma of the lower lip which was regarded as inoperable by the surgeons, and referred to the dermatological clinic. Apart from the pigmentation the skin presented the usual features of this disease. It was thin, finely scaly, red, in places presenting a cicatricial aspect, and showed numerous stellar and punctate telangiectases. Microscopic examination of the skin revealed a diffuse atrophy of the epithelium, infiltration with lymphocytes, disappearance of the elastin, severe congestion, and fragmentation of the connective-tissue fibers in the superficial portions of the derma, and hypertrophy of the elastin in the deep layers. The epithelioma was of the spinocellular variety, and was accompanied by enlargement of the submaxillary glands. Under the influence of radiotherapy there was a notable diminution in its size and the glands became smaller and more movable. In the author's opinion this case proves that the atrophic condition of the skin is the one essential feature of this malady; all the other phenomena may be wanting, epithelioma being only a complication. In his opinion the malady ought to be classed among the congenital dyskeratoses.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Significance of Streptococci and Leukocytes in Milk.—In the author's abstract of a paper presented at the annual meeting of the American Public Health Association in the city of Mexico, in December, 1906, NORMAN McLEOD HARRIS states that cultural and biological tests have failed to differentiate pathogenic from non-pathogenic forms of milk streptococci, although it is thought that milk obtained from cows suffering from acute mammitis is responsible for epidemics of severe sore-throat, tonsillitis, and gastro-intestinal disturbances in man. Hence, it would seem that in the absence of any such outbreaks the cocci are ordinarily to be considered as of no significance. With reference to the so-called pus cells in milk, we are at a disadvantage in having little definite information about normal leukocytosis. Concerning this point the observations of H. L. Russell and Conrad Hoffman (author's abstract—Leukocyte Content of Milk Drawn from Apparently Healthy Animals) are of value. From an examination of the several existing standards it is evident that there is a wide range in the limits which are recognized as indicating the presence of pus in milk and that data are altogether lacking to warrant the formulation of a proper scientific standard. They have examined milk from cows with no discoverable udder lesions or previous history of garget, and have found very wide variations, some specimens which are wholly satisfactory from a clinical standpoint yielding counts much in excess of accepted standards. They present data showing that "physiological leukocytosis may manifest itself in the milk of animals subjected to environmental conditions such as can in no material way affect the wholesomeness of the milk supply," and they emphasize the necessity of a thorough study of the subject before excluding from the market milk from cows in which no moribd processes can be detected.

W. RULLMANN and R. TROMSDORFF (*Arch. f. Hyg.*, 1906, lix, 224) confirm Bergey's statement that a large number of leukocytes in milk means a large content of streptococci, and they assert that the latter is always a consequence of mastitis, which disease appears to spread in some way unknown. They examined cows, which, as far as clinical evidence was concerned, were perfectly sound, and found that the milk of 19.5 per cent. thereof contained as much as 1 per cent. by volume of leukocytes. Doubtless, infection is spread by the milkers, and since mastitis makes milk dangerous and diminishes its yield, the strictest cleanliness on the part of the milker is necessary. The streptococci of the udder are especially dangerous to infants and young children.

According to P. G. HEINEMANN (author's abstract, The Pathogenicity of Streptococcus lacticus), Streptococcus lacticus is closely related to Streptococcus pyogenes, not only morphologically and culturally, but also in pathogenic property. Its virulence is increased by repeated passages through rabbits, so that after five or more passages 2 c.c. of a twenty-four-hour-old culutre produces death. The lesions produced in rabbilts are the same in kind and extent as those produced by Streptococcus pyogenes obtained from pathological conditions in human beings.

Infantile Mortality and Goats' Milk.—The superiority of the goat over the cow in the matter of cleanliness (difference in character of excreta, disinclination to lie down in filth); its immunity to tuberculosis, as shown by the fact that not a single case of the disease has been observed among the hundreds of thousands of goats slaughtered at La Villette, in Paris; the greater digestibility of the curd, which is flocculent rather than hard, leads WILLIAM WRIGHT (*Lancet*, November 3, 1906) to advocate the use of goats' milk in place of cows' milk in infant feeding. He asserts that it is superior in nutriment and more suitable in every way; that its proportionate cost of production is far less; that the proportionate yield is far greater; and that if the animal is properly fed and restrained, the milk will have no odor.

Frequency of Tuberculosis in Dairies Supplying Paris.—According to H. MARTEL (*Revue de la Soc. scientif. d'hyg. aliment.*, 1906, ii, 559) of 628 cows whose milk constitutes part of the supply of Paris, 215, or 42.51 per cent., reacted to tuberculin. To avoid contagion at the cow-stable, he recommends the tuberculin test to prevent the introduction of diseased animals into a herd and periodical testing to discover and eliminate infected animals therefrom.

Cleanliness versus Pasteurization of Milk.—Concerning the comparative value of pasteurization of milk produced under ordinary conditions and of sterilization of the milk utensils and general cleanliness of production the experience of the Board of Health of Rochester, New York, as related by its Health Officer, GEORGE W. GOLER (*Arch. of Ped.*, September, 1906) is of great interest. During the quinquennium 1895 to 1900, children brought to the milk station were fed on pasteurized milk, and during the next same period they were given raw milk produced under sanitary conditions. In spite of increase in population due to natural increase and annexation, there were fewer deaths of children under five years of age during the latter period.

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